A DISSERTATION ON

"COMPARATIVE STUDY OF ORBITAL DOPPLER PARAMETERS IN DIABETICS WITH RETINOPATHY AND DIABETICS/ HEALTHY CONTROLS WITHOUT RETINOPATHY"

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CERTIFICATE

This is to certify that the dissertation titled **"COMPARATIVE STUDY OF ORBITAL DOPPLER PARAMETERS IN DIABETICS WITH RETINOPATHY AND DIABETICS/ HEALTHY CONTROLS WITHOUT RETINOPATHY"** submitted by **Dr.M.ThangaMeena,** appearing for **M.D.RADIODIAGNOSIS** degree examination in May 2019, is a bonafide record of work done by her under my guidance and supervision in partial fulfillment of requirements of The Tamilnadu Dr. M.G.R Medical University, Chennai. I forward this to The Tamilnadu Dr. M.G.R Medical University, Chennai.

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DECLARATION

I, **Dr. M.ThangaMeena,** certainly declare that this dissertation titled, "**COMPARATIVE STUDY OF ORBITAL DOPPLER PARAMETERS IN DIABETICS WITHRETINOPATHY AND DIABETICS/ HEALTHY CONTROLS WITHOUT RETINOPATHY**", represent a genuine work of mine, done at the Barnard Institute of Radiology, Madras Medical College and Government General Hospital, under the supervision of **Prof.D.RAMESH, M.D.R.D**., Professor, Barnard Institute of Radiology, Madras Medical College and Rajiv Gandhi Government General Hospital.

I, also affirm that this bonafide work or part of this work was not submitted by me or any others for any award, degree or diploma to any other university board, neither in India or abroad. This is submitted to The Tamil Nadu Dr.MGR Medical University, Chennai in partial fulfilment of the rules and regulation for the award of Master of Radiodiagnosis Branch VIII

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Dr.M.Thanga Meena

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COMPARATIVE STUDY OF ORBITAL DOPPLER PARAMETERS IN DIABETICS WITH RETINOPATHY AND DIABETICS/ HEALTHY CONTROLS WITHOUT RETINOPATHY

1.INTRODUCTION

Diabetes mellitus is one of the most common non communicable diseases globally. The prevalence of diabetes in our country is increasing. As the prevalence of diabetes is rising, the systemic complications that include retinopathy, nephropathy and neuropathy and involvement of cardiovascular system are also increasing. Diabetic retinopathy is a vascular disorder affecting the microvasculature of retina caused by changes in the retinal blood vessels. If untreated, it may lead to blindness. Therefore, if diagnosed and treated promptly, blindness is usually preventable. Color Doppler imaging is a new method that enables us to assess the orbital vasculature. It allows for simultaneous two dimensional anatomical and Doppler evaluation of hemodynamic characteristics of retinal artery [1].

For diagnosis of early changes in retinal blood flow in diabetes mellitus without retinopathy duplex color Doppler ultrasonography is the investigation of choice to assess the problem very quickly without any invasive procedures. Thus, this would be a beneficial tool for the ophthalmologist to assess and make quick decision about patient with impaired blood flow and to take necessary actions. They enable qualitative and quantitative assessment of the ophthalmic artery (OA) and its branches [2].

The most frequently assessed vessels include: the ophthalmic artery, central retinal artery and central retinal vein. During examinations, vascular morphology is assessed and blood flow parameters are evaluated, such as: peak systolic velocity (PSV), end-diastolic velocity (EDV), resistance index (RI) and pulsatility index (PI). The measurements are taken with the use of a linear or sector probe with the frequency of over 7 MHz by applying it to the closed eyelid of a patient in the supine position. Pressure to the eyeball should be avoided. It is recommended to maintain uniform conditions of examinations, such as lighting in the room, size of the gate and a constant site for measurements in subsequent examinations [3].

2.RATIONALE OF THE STUDY

Diabetic retinopathy (DR) is one of the leading causes of preventable blindness in India and worldwide. Annual retinal examinations are mandatory for all diabetic patients for early detection of DR as it can be asymptomatic. One of the main motivations for screening for DR is established efficacy of laser photocoagulation in preventing visual loss[4].

International disease severity scale for DR by Wilkinson is now used. This proposes five levels for grading of DR, based on risk of progression. Classification is worded as follows: none, mild non proliferative diabetic retinopathy (NPDR), moderate NPDR, severe NPDR or proliferative diabetic retinopathy (PDR). Presence and severity of Diabetic Macular Edema (DME) by Wilkinson is classified separately. The progression from mild stages of DR to advanced stages occurs in a stepwise fashion. The earliest visible manifestation of DR is appearance of micro aneurysms in the retina. If glycemic control is inadequate, retinal vascular abnormalities like intraretinal hemorrhages and cotton wool spots can then occur. The clinical features of DR are summarized below. Signs of increasing ischemia are venous changes like beading and looping, intraretinal micro vascular abnormalities and increasing number of retinal hemorrhages and exudation. When these signs progress beyond certain defined thresholds, severe NPDR is diagnosed. The level of severe NPDR carries with it the most risk for progression to PDR[4].

Diabetic retinopathy is a vascular disorder affecting the microvasculature of retina.If untreated, it may lead to blindness which is usually preventable. Color Doppler imaging is a new method that enables us to assess the orbital vasculature with two dimensional anatomical and Doppler evaluations of hemodynamic characteristics of retinal artery. It is a useful tool when standard diagnostic procedures are difficult in the presence of cataract or hazy media.

The utility of the doppler in south Indian popoulation has not been studied extensively.Thus our present study is planned to compare the orbital doppler parameters in diabetics with retinopathy and diabetics/ healthy controls without retinopathy in South Indian population and assess the utility of the Orbital Doppler study in Diabetic retinopathy.

3.REVIEW OF LITERATURE

Embryology: The eyes begin to develop in 22-day embryo when **optic grooves** appear. The eyes are derived from four sources:

- **Neuroectoderm** of the forebrain
- **Surface ectoderm** of the head
- **Mesoderm** between the previous two layers
- **Neural crest cells**

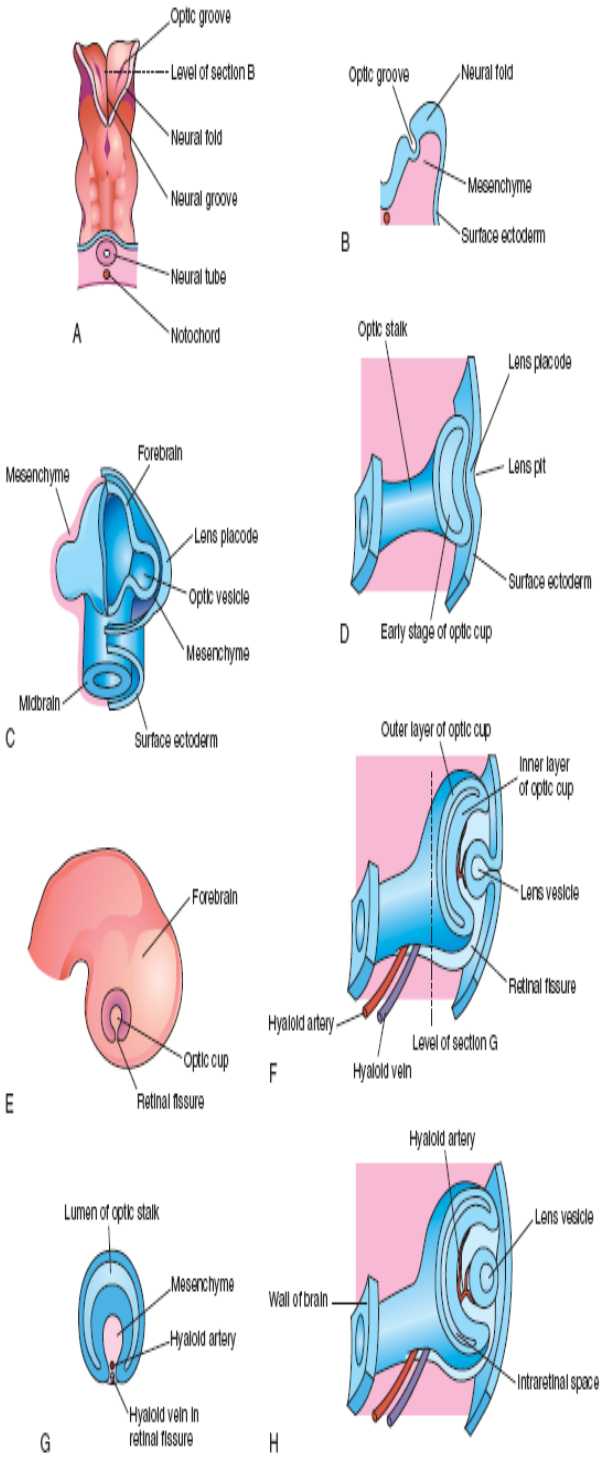
The **neuroectoderm** differentiates into the retina, posterior layers of iris, and optic nerve. The **surface ectoderm** forms the lens of the eye and the corneal epithelium. The **mesoderm** between the Neuroectoderm and surface ectoderm gives rise to the fibrous and vascular coats of the eye. **Neural crest cells** migrate into the mesenchyme and differentiate into the choroid, sclera, and corneal endothelium.

The first evidence of eye development is the appearance of **optic grooves** in the neural folds at the cranial end of the embryo .As the **neural folds** fuse to form the forebrain, the optic grooves evaginated (protruded) from the future diencephalon to form hollow diverticula (outpocketings) called **optic vesicles,** which project from the wall of the forebrain into the adjacent mesenchyme. The vesicles soon come in contact with the surface ectoderm. As the optic vesicles grow, their distal ends expand and their connections with the forebrain constrict to form hollow **optic stalks**. Concurrently, the surface ectoderm adjacent to the vesicles thickens to form **lens placodes**, which are the primordia of the lenses.

The lens placodes invaginate as they sink deep to the surface ectoderm, forming **lens pits** The edges of the lens pits approach each other and fuse to form spherical **lens vesicles**, which gradually lose their connection with the surface ectoderm. As the lens vesicles are developing, the optic vesicles invaginate to form double-walled **optic cups**, which consist of two layers that are connected to the developing brain by optic stalks. The optic cup becomes the retina, and the optic stalk becomes the optic nerve. The lens and part of the cornea develop from the ectoderm and mesoderm.

Linear grooves **(retinal fissures)** develop on the ventral surface of the optic cups and along the optic stalks. The center of the optic cup, where the retinal fissure is deepest, forms the **optic disc**, where the neural retina is continuous with the optic stalk. The developing **axons of the ganglion cells** pass directly into the optic stalk and convert it into the optic nerve. The retinal fissures contain vascular mesenchyme from which **hyaloid blood vessels** develop. The **hyaloid artery**, a branch of the ophthalmic artery supplies the inner layer of the optic cup, the lens vesicle and the mesenchyme in the **cavity of the optic cup**. The **hyaloid vein** returns blood from these structures. As the edges of the retinal fissure fuse, the **hyaloid vessels** are enclosed within the **primordial optic nerve**. Distal parts of the hyaloid vessels eventually degenerate, but proximal parts of them persist as the **central artery** and **vein of retina.**

Fig.3.1Early stages of eye development



A, Dorsal view of the cranial end of an embryo at approximately 22 days shows the optic grooves, which are the first indication of eye development.

B, Transverse section of a neural fold shows the optic groove in it.

C, Schematic drawing of the forebrain of an embryo at approximately 28 days shows its covering layers of mesenchyme and surface ectoderm.

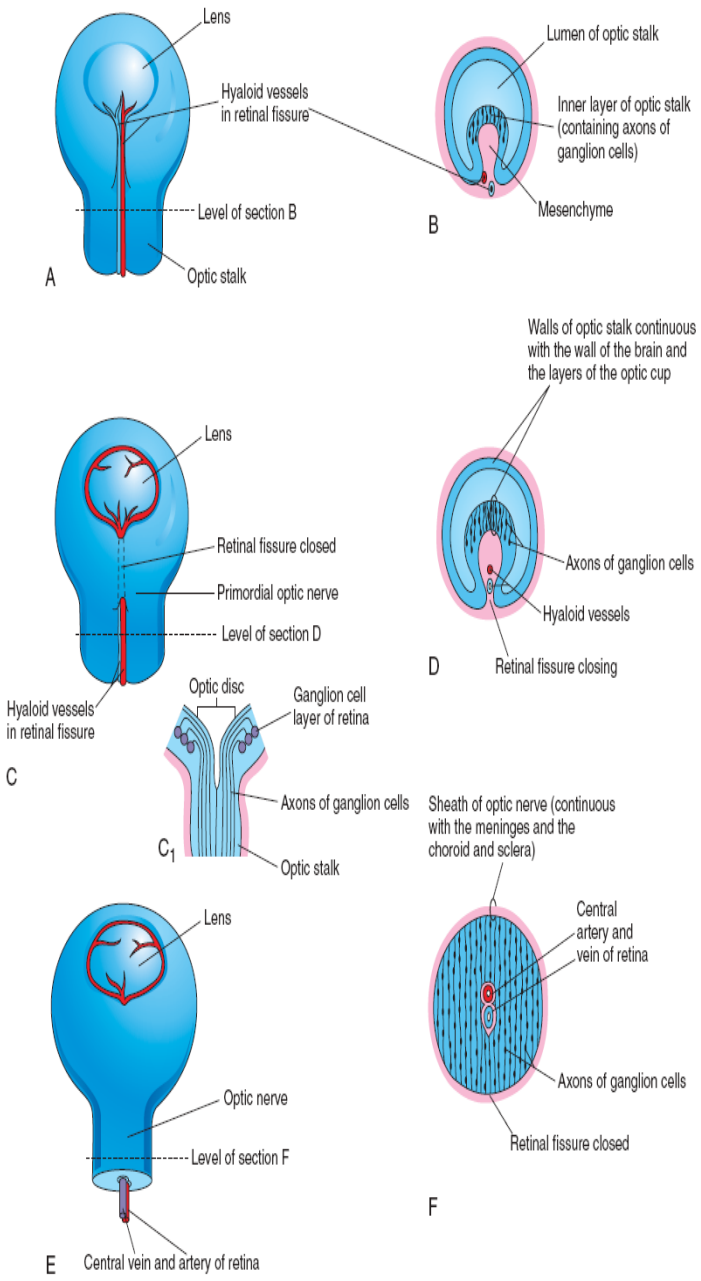
D, F, and H, Schematic sections of the developing eye show the successive stages in the development of the optic cup and lens vesicle.

E, Lateral view of the brain of an embryo at approximately 32 days shows the external appearance of the optic cup.

G, Transverse section of the optic stalk shows the retinal fissure and its contents. The edges of the retinal fissure are growing together, thereby completing the optic cup and enclosing the central artery and vein of the retina in the optic stalk and cup.

Courtesy: THE DEVELOPING HUMAN, CLINICALLY ORIENTED EMBRYOLOGY

Fig.3.2: Closure of the retinal fissure and formation of the optic nerve.



A, C, and E, Views of the inferior surface of the optic cup and stalk show progressive stages in the closure of the retinal fissure.

C1, Schematic drawing of a longitudinal section of a part of the optic cup and stalk shows the optic disc and axons of ganglion cells of the retina growing through the optic stalk to the brain.

B, D, and F, Transverse sections of the optic stalk show successive stages in the closure of the retinal fissure and formation of the optic nerve.

Courtesy: THE DEVELOPING HUMAN, CLINICALLY ORIENTED EMBRYOLOGY

The lumen of the optic stalk is gradually obliterated as axons of ganglion cells accumulate in the inner layer of the optic stalk as the optic nerve forms[5].

Anatomy of eyeball and its blood supply:

Eyeball

Each eyeball is a cystic structure kept distended by the pressure inside it.

• Shape. Although, generally referred to as a globe, the eyeball is not a sphere but an oblate spheroid.

• Poles. The central point on the maximal convexities of the anterior and posterior curvatures of the eyeball is called the anterior and posterior pole, respectively.

• Equator of the eyeball lies at the mid plane between the two poles

Coats of the eyeball

The eyeball comprises three coats: outer (fibrous coat), middle (vascular coat) and inner (nervous coat).

1. Fibrous coat. It is a dense strong wall which protects the intraocular contents. Anterior 1/6th of this fibrous coat is transparent and is called cornea.

Posterior 5/6th opaque part is called sclera. Cornea is set into the sclera like a watch glass. Junction of the cornea and sclera is called limbus. Conjunctiva is firmly attached at the limbus.

2. Vascular coat (uveal tissue). It supplies nutrition to the various structures of the eyeball. It consists of three parts, from anterior to posterior, which are: iris, ciliary body and choroid.

3. Nervous coat (retina). It is concerned with visual functions and projects to visual cortex through the visual pathway.

Segments and chambers of the eyeball

The eyeball can be divided into two segments: anterior and posterior.

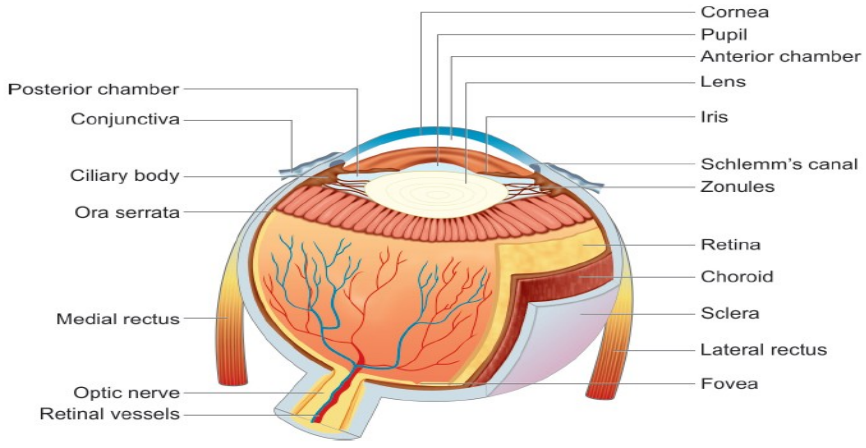
1. Anterior segment: It includes crystalline lens (which is suspended from the ciliary body by zonules), and structures anterior to it, viz., iris, cornea and two aqueous humour-filled spaces: anterior and posterior chambers.

■Anterior chamber: It is bounded anteriorly by the back of cornea, and posteriorly by the anterior surface of iris and part of ciliary body. The anterior chamber is about 2.5 mm deep and is slightly shallower in hypermetropes and deeper in myopes.

■Posterior chamber: It is a triangular space containing 0.06 ml of aqueous humour. It is bounded anteriorly by the posterior surface of iris and part of ciliary body, posteriorly by the crystalline lens and its zonules, and laterally by the ciliary body.

2. Posterior segment. It includes the structures posterior to lens, viz., vitreous humour, retina, choroid and optic disc[6] .

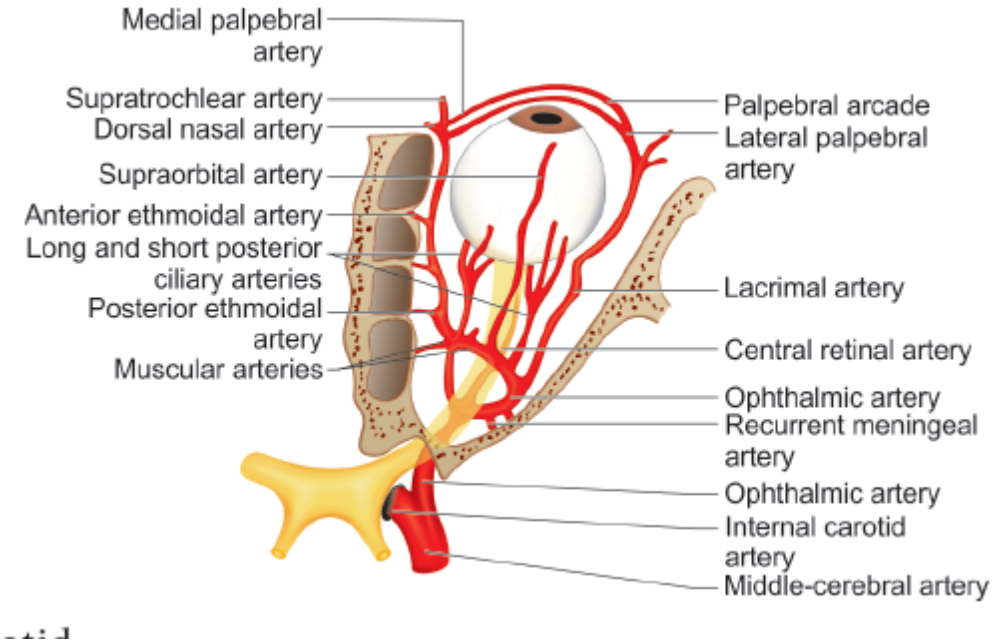
Fig.3.3: Gross anatomy of the eyeball



Courtesy: Comprehensive ophthalmology 6th edition, Khurana

Vascular supply and drainage:

The arterial supply to the eye is provided by several branches from the ophthalmic artery, which is derived from the internal carotid artery .These branches include the central retinal artery, the short and long posterior ciliary arteries, and the anterior ciliary arteries. Venous outflow from the eye is primarily via the vortex veins and the central retinal vein, which merge with the superior and inferior ophthalmic veins that drain into the cavernous sinus, the pterygoid venous plexus and the facial vein[7]



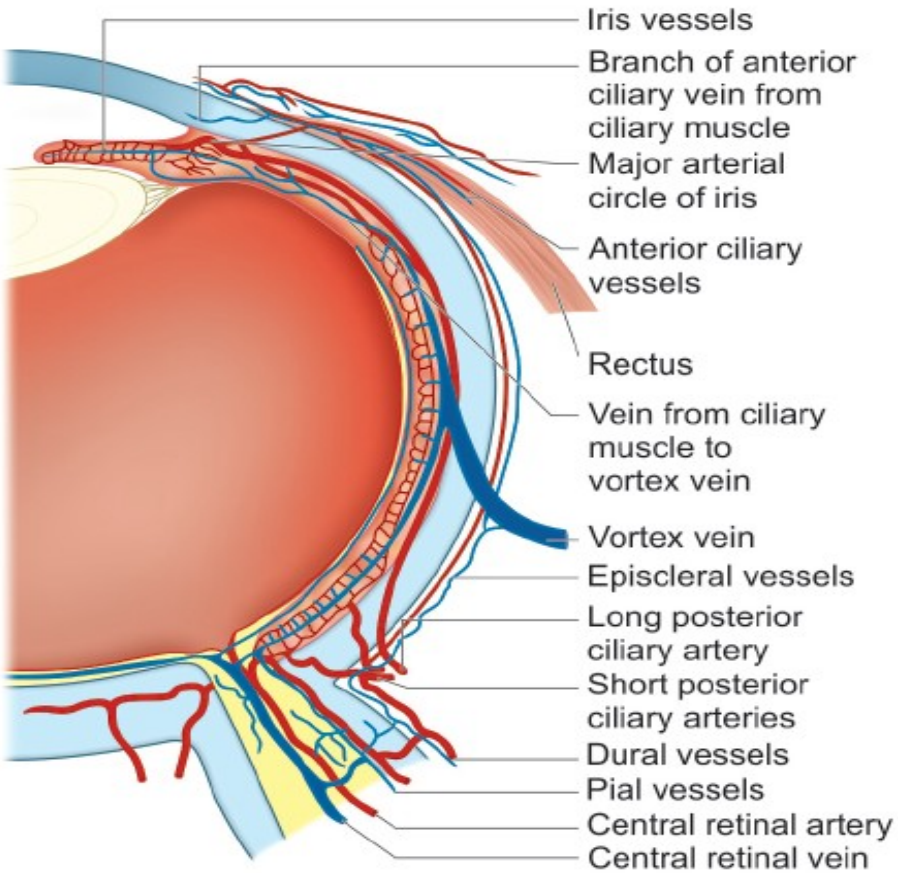
Courtesy: Comprehensive ophthalmology 6th edition, Khurana

Fig.3.4: Ophthalmic artery and its branches

The OA emerges through the optic foramen in the posterior orbit just lateral to the optic nerve. More anteriorly, the OA usually crosses over the optic nerve and continues into the anteromedial orbit. The central retinal, long and short ciliary, lacrimal, supraorbital, and

supratrochlear arteries are all branches of the OA that can be visualized in most normal orbits. The OA can be consistently located with color flow imaging by scanning medial to the optic nerve approximately 15 mm posterior to the globe. The central retinal artery (CRA) and central retinal vein (CRV) nourish the inner two-thirds of the retina. The artery and vein parallel one another in the center of the distal optic nerve [8].

Fig.3.5: Blood supply of eyeball



Courtesy: Comprehensive ophthalmology 6th edition, Khurana

Diabetes Mellitus:

Diabetes mellitus comprises of a group of chronic metabolic disorders involving the principal metabolic fuels, carbohydrates, fats as well as proteins. The disorder results from absolute or relative deficiency in insulin secretion often along with defect in insulin action. The syndrome of diabetes mellitus is characterized by chronic hyperglycemia (blood glucose above defined limits) with or without glucosuria and a tendency to develop ketoacidosis. Polyuria, polydipsia, asthenia and weight loss, the cardinal symptoms of severe hyperglycemia, may not be present in a proportion of cases. There is an increased susceptibility to bacterial and fungal infection. In course of time, prolonged hyperglycemia and associated metabolic aberrations result in tissue toxicity manifested as accelerated atherosclerosis, renoretinal microangiopathy and neuropathy leading to a variety of vascular, neurological and focal complications[9].

Epidemiology:

Currently Diabetes Mellitus has emerged as a challenge worldwide in epidemic proportions. According to the WHO estimates, by the year 2030, the number of people affected with DM will increase to 360 million [1]. Changes in life style, dietary pattern, physical inactivity and obesity are responsible for increasing the prevalence of diabetes mellitus into the developing countries which is no longer a disease of developed countries. All DM patients are at risk of developing diabetic retinopathy (DR)[10].

India, a country experiencing rapid socioeconomic progress and urbanization, carries a considerable share of the global diabetes burden. Studies in different parts of India have demonstrated an escalating prevalence of diabetes not only in urban populations, but also in rural populations as a result of the urbanization of lifestyle parameters. The prevalence of prediabetes is also high. Recent studies have shown a rapid conversion of impaired glucose tolerance to diabetes in the southern states of India, where the prevalence of diabetes among adults has reached approximately 20% in urban populations and approximately 10% in rural populations. Because of the considerable disparity in the availability and affordability of diabetes care, as well as low awareness of the disease, the glycemic outcome in treated patients is far from ideal. Lower age at onset and a lack of good glycemic control are likely to increase the occurrence of vascular complications. The economic burden of treating diabetes and its complications is considerable[11].

Diagnosis:

Blood glucose is normally maintained in a very narrow range, usually 70 to 120 mg/dL. According to American Diabetes Association (ADA) and the World Health Organization (WHO), **diagnostic criteria for diabetes include the following:**

1. A fasting plasma glucose greater than or equal to 126 mg/dL, and/or

2. A random plasma glucose greater than or equal to 200 mg/dL (in a patient with classic hyperglycemic signs), and/or

3. A 2-hour plasma glucose greater than or equal to 200 mg/dL during an oral glucose tolerance test with a loading dose of 75 gm, and/or

4. A glycated hemoglobin (HbA1c) level greater than or equal to 6.5%

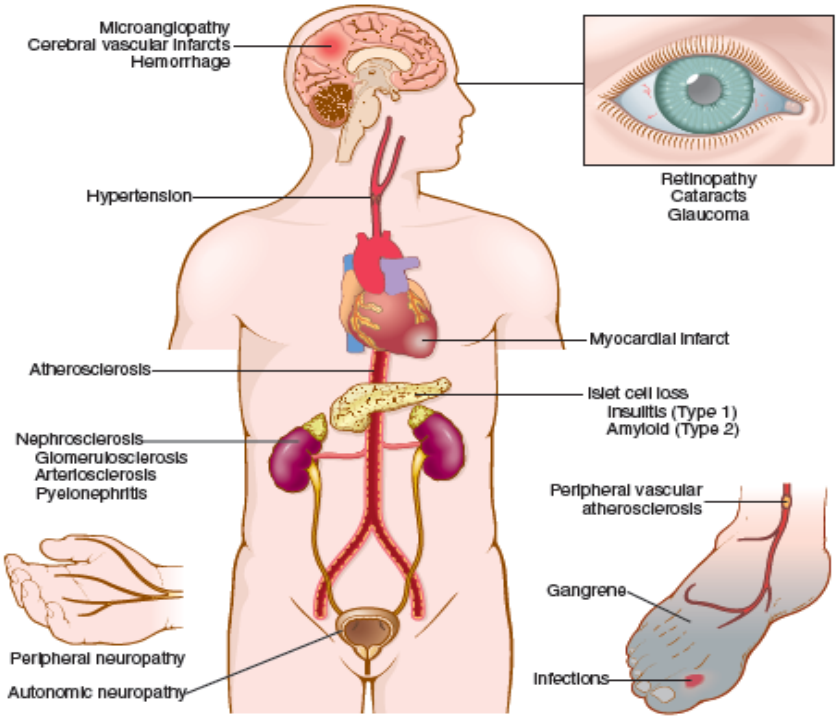
All tests, except the random blood glucose test in a patient with classic hyperglycemic signs, need to be repeated and confirmed on a separate day. Of note, many acute conditions associated with stress, such as severe infections, burns, or trauma, can lead to transient hyperglycemia due to secretion of hormones such as catecholamines and cortisol that oppose the effects of insulin. The diagnosis of diabetes requires persistence of hyperglycemia following resolution of the acute illness.

Impaired glucose tolerance (prediabetes) is defined as:

1. A fasting plasma glucose between 100 and 125 mg/dL (impaired fasting glucose) and/or
2. A 2-hour plasma glucose between 140 and 199 mg/dL during an oral glucose tolerance test and/or
3. HbA1c level between 5.7% and 6.4% [12].

Complications:

The complications related to diabetes include acute, life-threatening metabolic disturbances and chronic complications which predominantly involve small and large blood vessels and the nervous system. The risk of vascular complications increases with the duration of the disease. Diabetic retinopathy is the most common microangiopathic complication, specific for type I and II diabetes[13].



Courtesy: Robbins Pathology, 10th edition

Fig.3.6: Long term complications of Diabetes Mellitus

Pathogenesis of Diabetic microangiopathy:

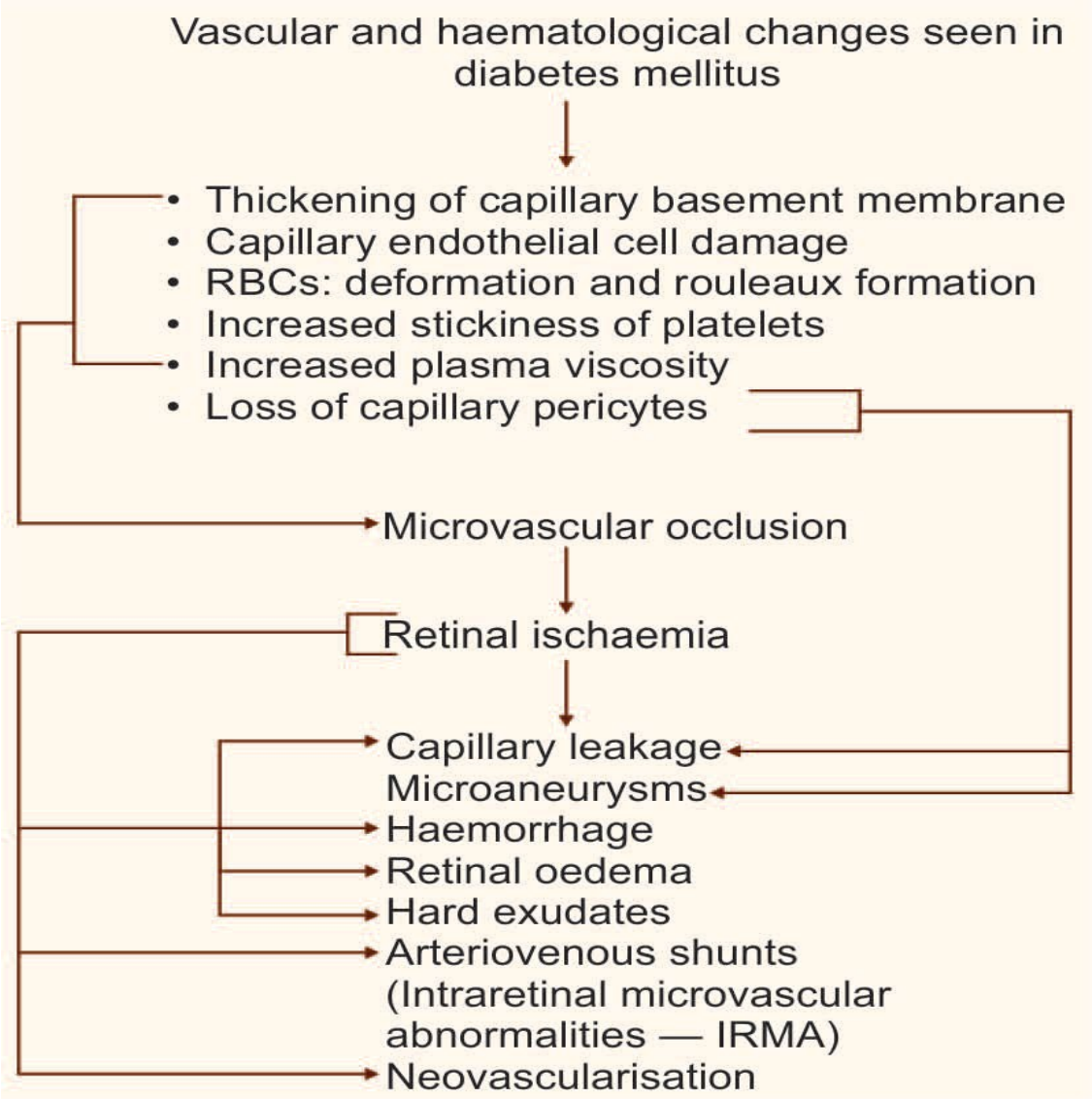
One of the most consistent morphologic features of diabetes is **diffuse thickening of basement membranes.** The thickening is most evident in the capillaries of the skin, skeletal muscle, retina, renal glomeruli, and renal medulla. However, it also may be seen in nonvascular structures such as renal tubules, the Bowman capsule, peripheral nerves, and placenta. By both light and electron microscopy, the basal lamina separating parenchymal or endothelial cells from the surrounding tissue is markedly thickened by concentric layers of hyaline material composed predominantly of type IV collagen. Of note, despite the increase in the thickness of basement membranes, diabetic capillaries are

leaky, leading to extravasation of plasma proteins. The microangiopathy underlies the development of diabetic nephropathy, retinopathy and some forms of neuropathy. The ocular involvement may take the form of retinopathy, cataract formation, or glaucoma. Retinopathy, the most common pattern, consists of a constellation of changes that together are considered by many ophthalmologists to be virtually diagnostic of the disease. The lesion in the retina takes two forms: non proliferative retinopathy and proliferative retinopathy.

Non proliferative retinopathy includes intraretinal or preretinal hemorrhages, retinal exudates, micro aneurysms, venous dilations, edema, and, most importantly, thickening of the retinal capillaries (microangiopathy). The retinal exudates can be "soft" (micro infarcts) or "hard" (deposits of plasma proteins and lipids). The micro aneurysms are discrete saccular dilatations of retinal choroidal capillaries that appear through the ophthalmoscope as small red dots. Dilatations tend to occur at focal points of weakening, resulting from loss of pericytes. Retinal edema presumably results from excessive capillary permeability. Underlying all of these changes is the microangiopathy, which is

thought to lead to loss of capillary pericytes and hence to focal weakening of capillary structure

Proliferative retinopathy is a process of neovascularization and fibrosis. This lesion leads to serious consequences, including blindness, especially if it involves the macula. Vitreous hemorrhages may result from the rupture of newly formed capillaries; the subsequent organization of the hemorrhage can pull the retina off its substratum (retinal detachment) [12].



Courtesy: Comprehensive Opthalmology 6th edition ,Khurana

Fig.3.7: Flowchart depicting pathogenesis of diabetic retinopathy

Risk factors

1. Duration of diabetes is the most important determining factor. After 10 years, 20% of type I and 25% of type II diabetics develop retinopathy. After 20 years, 90% of type I and 60% of type II diabetics develop retinopathy. After 30 years, 95% of both type I and type II diabetics develop retinopathy.

2. Age of onset of diabetes also act as a risk factor. The risk of retinopathy in a child with onset of diabetes at the age of 2 years is negligible for the first 10 years. After onset of puberty, age of onset is not a risk factor.

3. Sex. Incidence is more in females than males (4:3).

4. Poor metabolic control is less important than duration, but is nevertheless relevant to the development and progression of DR.

5. Heredity. It is transmitted as a recessive trait without sex linkage. The effect of heredity is more on the proliferative retinopathy.

6. Pregnancy may accelerate the changes of diabetic retinopathy.

7. Hypertension, when associated, may also accentuate the changes of diabetic retinopathy.

8. Other risk factors include smoking, obesity, anemia and hyperlipidemia [14].

CLASSIFICATION AND CLINICAL FEATURES OF DIABETIC RETINOPATHY

The Early Treatment Diabetic Retinopathy Study severity scale based on modified Airlie House classification of DR is the gold standard for grading DR [15]. It is based on seven fields stereo color photographs. This grading system has been used for several landmark clinical trials. The ETDRS classification is difficult to use in clinical settings as it is time consuming requires correlations with standard photographs and needs skilled photographers and trained graders. Hence, International disease severity scale for DR by Wilkinson is now used [16].This proposes five levels for grading of DR, based on risk of progression.

Classification is worded as follows:

A. None

B. Mild non proliferative diabetic retinopathy

C. Moderate non proliferative diabetic retinopathy

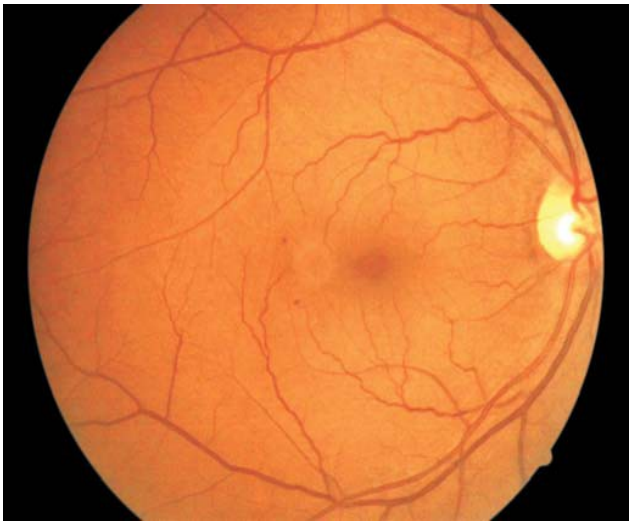
D. Severe non proliferative diabetic retinopathy

E. Proliferative diabetic retinopathy

Table.3.1:International disease severity scale for diabetic retinopathy by Wilkinson

Retinopathy stage	*Findings on ophthalmoscopy*
No apparent retinopathy	No abnormalities
Minimal NPDR	Microaneurysms only
Mild to Moderate NPDR	More than just microaneurysms but less than severe NPDR
Severe NPDR	Any of the following: • More than 20 intraretinal hemorrhages in each of the four quadrants • Definite venous beading in two or more quadrants. • Prominent intraretinal microvascular abnormalities in one or more quadrant AND • No signs of proliferative retinopathy
Proliferative DR	One of the following: • Neovascularization • Vitreous or preretinal hemorrhage

Fig.3.8: Mild non-proliferative diabetic retinopathy with microaneurysms



Courtesy: RSSDI TEXTBOOK OF DIABETES MELLITUS

Fig.3.9: Moderate non-proliferative diabetic retinopathy with microaneurysms and cotton wool spots

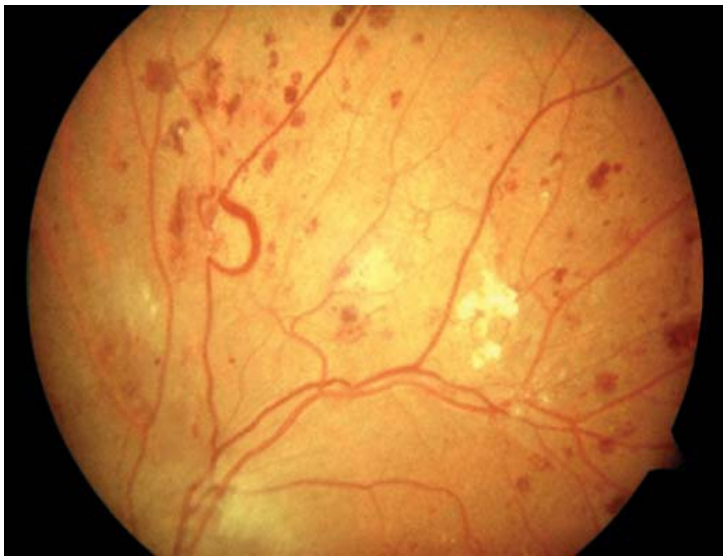


Fig.3.10: Severe non-proliferative diabetic retinopathy with venous looping and blot hemorrhages

Fig.3.11:Proliferative diabetic retinopathy

Table.3.2:Presence and severity of DME by Wilkinson is classified separately.

Macular edema	
Absent	No retinal thickening or hard exudates in posterior pole
Present	*Mild DME*: some retinal thickening or hard exudates in posterior pole but distant from the macula *Moderate DME*: retinal thickening or hard exudates approaching the centre of the macula but not involving the centre *Severe DME*: retinal thickening or hard exudates involving the centre of macula.

(DME: Diabetic macular edema)

Courtesy: RSSDI TEXTBOOK OF DIABETES MELLITUS

Fig.3.12: Mild diabetic macular edema

The progression from mild stages of DR to advanced stages occurs in a stepwise fashion [17].The earliest visible manifestation of DR is appearance of microaneurysms in the retina. If glycemic control is inadequate, retinal vascular abnormalities like intraretinal hemorrhages and cotton wool spots can then occur.

The clinical signs of DR are summarized below. Signs of increasing ischemia are venous changes like beading and looping, intraretinal micro vascular abnormalities and increasing number of retinal hemorrhages and exudation. When these signs progress beyond certain defined thresholds, severe NPDR is diagnosed [17].The level of severe NPDR carries with it the most risk for progression to PDR.

Table.3.3: clinical signs of diabetic retinopathy

Type of lesion	*Pathogenesis*	*Sign*	*Significance*
Microaneurysms	Saccular outpouchings of the retinal capillary wall Due to pericyte loss	Tiny red dots in the macula and different quadrants of the retina	Earliest sign of diabetic retinopathy
Retinal hemor-rhages			
(a) Flame shaped hemorrhages	Arises from precapillary arterioles	Occur in the superficial retinal nerve fibre layer	
(b) Dot and blot shaped hemor-rhages	From venous end of capillaries	From middle layers of retina	Capillary occlusion and retinal ischemia
Hard exudates	From chronic localized retinal edema	Waxy yellow lesions Can be powdery, plaque or as circinate rings	Composed of lipoprotein and lipid filled macrophages within outer plexiform layer. Vision threatening if located at fovea.
Cotton wool spots	Nerve fibre layer infarcts.	White fluffy superficial lesions	Sign of retinal ischemia
Intraretinal microvascular abnormalities	A-V shunts at areas of capillary closure	Fine irregular segments of capillaries between arterioles to venules	Indicates severe retinal ischemia. increased likelihood of progression to proliferative DR
Venous changes	Due to capillary occlusion and retinal ischemia	Beading, looping, due to venous dilatation	Indicative of severe retinal ischemia
Neovasculari-zation	Retinal ischemia induces angiogenic growth factors which causes abnormal new retinal vessels formation	Can be seen in the quadrants of retina (new vessels elsewhere) or on the optic nerve head (new vessels at disk)	Results in preretinal, vitreous hemorrhage, tractional retinal detachment

Courtesy: RSSDI textbook of diabetes mellitus

The following methods are used to assess diabetic retinopathy: ophthalmoscopic evaluation of the fundus, examination through a slit lamp with the application of lenses, retinal photography, and supplemental examinations, such as: fluorescein angiography, optical coherence tomography of the retina and B-mode ultrasound imaging [18,19].

Direct ophthalmoscopy:

It produces high magnification (15×) and is very useful in identifying microaneurysms, retinal hemorrhages, hard exudates, cotton wool spots and new vessels.

Indirect ophthalmoscopy:

It aids to assess the periphery of the fundus.

Slit lamp biomicroscopy with contact lens:

It is very helpful to assess Diabetic macular edema (DME). It is important to detect DME in the assessment of DR as this is the most frequent cause of decreased vision from retinopathy.

Retinal Color Photography:

It is useful to document retinal lesions. It is more reproducible than clinical examination and useful in research studies. It helps to document progression of DR and response of treatment.

Fundus Fluorescein Angiography (FFA):

It involves photographic surveillance of passage of fluorescein through the retinal and choroidal circulation following intravenous injection. 5 ml of 10% fluorescein dye is injected into the vein and after an arm retina time of 11–15 seconds and serial angiogram pictures at different phases are taken using digital retinal camera. FFA is indicated in patients with DR to assess areas of leakage and also for macular ischemia (where laser has to be avoided). In severe NPDR, it helps to assess risk of developing PDR. Patients with unexplained visual loss will also be benefited with FFA [20].

Optical Coherence Tomography (OCT):

It is a non-invasive, non-contact imaging system which provides high resolution cross sectional images of retina and optic nerve. OCT uses light instead of sound waves. It is indicated to quantify the retinal thickness in the different areas of the macula, identify cystic spaces in the macula, identify macular holes, neurosensory detachment

and vitreomacular traction. It helps to assess the response of treatment like laser and intravitreal injections and in analysis of optic nerve head and retinal nerve fiber layer [21].

B-Scan Ultrasound:

It helps to assess the presence of vitreous hemorrhage and retinal detachment in diabetic patients with opaque media.

Colour Doppler Imaging Of Orbital vessels:

Colour Doppler imaging (CDI) is a useful diagnostic tool in evaluating ocular and orbital diseases. Based on its capacity to combine two-dimensional B-mode image and functional blood flow analysis, this non-invasive method is suitable to study vascular abnormalities involving the central retinal artery, central retinal vein, posterior ciliary arteries, ophthalmic artery, and superior ophthalmic vein. It is also applied to assess hemodynamic changes in orbital vasculature. Furthermore, in comparison to other imaging methods such as computerized tomography (CT) and magnetic resonance imaging (MRI), CDI has the advantage of not using radiation. Ease of use and portability of equipment are also distinct advantages with CDI. Most importantly, CDI offers the capability of visualizing ocular and orbital vessels not frequently assessed by other methods [22].

Color Doppler imaging is a non-invasive method .The most frequently assessed vessels includes the ophthalmic artery, central retinal vein and artery, and the posterior ciliary arteries. The analysis of hemodynamic alterations in the retrobulbar vessels may deliver important information concerning circulation in diabetes and the changes observed in blood flow in the vessels with progression of diabetic retinopathy of the eyeball[3].

Colour Doppler Imaging: background and physical considerations

The Doppler effect is the property of sound waves or electromagnetic waves to undergo changes in their velocity of propagation by the movement of a given reflector. This is a physical principle first described by Johann Christian Doppler, an Austrian scientist in 1842[23]·Doppler ultrasonography is based on ultrasound wave reflection by a reflector (e.g. blood column), which moves at a given velocity in one direction in reference to a transducer. The frequency of the emitted sound is altered by the velocity of blood particles. This frequency is increased when the blood flow is moving towards the transducer and decreases when blood moves away. Calculation of the magnitude of reflected sound yields an estimate of blood flow; the faster the blood flow (reflector), the greater the difference between the reflected and emitted frequencies (Doppler shift – ΔF), as demonstrated by the formula,

$\Delta F = 2VF0 /C$, which can be rearranged to give:

$V = \Delta FC /2F0$ where, ΔF is frequency change, V is velocity of the reflector, F0 is sound source frequency, and C is the speed of sound in tissue .This equation is valid when transducer and reflector are parallel. Although orbital vessels are frequently parallel to the ultrasound beam, angle correction should be used when needed. Newer versions of the equipment automatically adjust for angle correction in the velocity calculations. Improvements in CDI techniques have tried to overcome limitations, such as angle dependence and difficulty in separating background noise from true flow in slow-flow states. Power Doppler sonography is able to evaluate low blood flow and has relative angle independence thereby offering superior depiction of tissue perfusion[24,25]·Power Doppler sonography may be particularly valuable in assessing small vasculature of the eye. Doppler information can be combined with gray-scale image ultrasound to provide

two-dimensional imaging (B-scan) and blood flow calculation (velocity and direction). Color duplex-scanning is obtained by representation of relative mean velocity of reflectors and emitters in a given area. Sound reflection signals are frequently displayed with color tone, saturation, and brightness as a function of their velocity. In addition, real-time blood flow assessment during the cardiac cycle is also possible (Doppler spectral analysis).

Table.3.4: Commonly used techniques in Doppler imaging.

Color Doppler	Velocity information is displayed as a color code and superimposed on top of a B-mode image
Continuous Doppler	Continuous generation of ultrasound waves coupled with continuous ultrasound reception performed by two different transducer heads (crystals) without bidimensional (2D) discrimination
Pulsed Doppler	Doppler information is sampled from a small sample volume (defined in 2D image), and presented on a timeline. It uses a transducer that alternates transmission and reception
Duplex scanning	A common name for the simultaneous presentation of 2D and Doppler information
Spectral analysis	Time × frequency diagram representative of blood flow in a cardiac cycle
Power Doppler	Improved technology in Doppler sonography that has the advantages of less direction dependence, higher sensitivity, and better contrast of vasculature

Courtesy: Opthalmic Ultrasonography, Elsevier

Examination technique and device parameters:

CDI examination of the globe and orbit is performed with the patient in the supine position, through closed eyelids. The transducer is placed on the eyelid using ophthalmic solution or acoustic gel. Care should be taken to avoid globe pressure that can lead to artifactual decrease in blood flow velocity. For ophthalmic applications, linear

transducers with sound frequencies ranging from 7.5 to 15 MHz are preferred. Horizontal and vertical grayscale imaging can be performed first in order to assess the entire ocular and orbital anatomy.

Color flow information is used to depict the major vascular structures in the orbit, which can be displayed in either blue or red. Color parameters can be subjectively assigned regarding the direction of the blood flow with respect to the transducer. In general, the flow moving towards the transducer is displayed in red, and away from it, is showed in blue.

Orbital vessels are frequently parallel to the sound beam, thus, for the majority of the time arteries are displayed in red and veins in blue. A pulsed Doppler spectral analysis is also obtained to distinguish between arterial and venous flow. CDI application in ophthalmology dates from the late 1980s. It is applied to evaluate several ocular and orbital diseases [26-38] .Several studies have demonstrated CDI velocities of orbital vessels in normal eyes with relatively good reproducibility [28, 29, 33, 35, 38-41].

TECHNIQUE:

Equipment:

A 7.5-MHz vector transducer with a 14-mm-wide face has an appropriate footprint and adequate depth of penetration for most examinations.

A 10-MHz probe gives better resolution in the near field and may be useful in evaluating abnormalities in the globe. A stand-off can be used to evaluate more anterior structures.

Examination Method:

Ultrasonogram (USG) examination of the orbit is performed through the closed eyelid with the patient supine. Contact lenses are removed prior to the examination. As little pressure as possible should be exerted on the globe to avoid inaccurate vascular recordings and patient discomfort; stabilizing the transducer with fingers on the forehead or cheek is helpful. The patient is instructed to keep the eyes closed and must be able to remain reasonably still for the examination.

Fig.3.13: USG technique. Photograph from a standard examination shows scanning being performed.

Sterile USG coupling gel has been applied to the eyelid. Gray-scale imaging is performed first to obtain an overview of the anatomy in the orbit. Imaging in several planes is possible. Colour flow information is then used to identify the major vascular structures in the orbit, as well as to assess the vascularity of any detected abnormalities. The standard examination takes approximately 10-15 minutes per eye. Color flow images were obtained. Pulsed Doppler recordings can then be obtained. A small Doppler gate is necessary to provide accurate and reproducible wave forms.

Angle correction was applied to the pulsed Doppler recordings to minimize errors in the measured velocities. The peak systolic (PSV) and end-diastolic (EDV) velocities are measured in the ophthalmic artery, central retinal artery, and in the central retinal vein. The measurements to calculate vascular resistance (expressed by the resistivity index and pulsatile index) using the following formula:

Resistivity index = (PSV-EDV)/PSV [42] and

Pulsatile index = (PSV-EDV)/Vmean

Where Vmean = 1/3 (PSV-EDV) +EDV [43], in all patients.

Signals from the ophthalmic artery can be located in the medial section of an eyeball, superior to the optic nerve, just lateral to and abutting the visible hypo echoic stripe representing the nerve. The central retinal artery originates from the ophthalmic artery and can be found anterior to the optic nerve, around 7.5 mm behind the ocular bulb.

A specific finding of the blood flow in the central retinal vein is its pulsatile character. As the velocity wave in a vein is not in direct relation to the cardiac systole and diastole, the blood flow parameters in this vessel are usually described as maximum (V max) and minimum blood velocity (Vmin) generally. The Velocity maximum and Velocity minimum in the central retinal vein is represented as the Peak systolic velocity and End diastolic velocity respectively.

Safety Issues:

There is several important safety issues related to color Doppler imaging of the orbit. The power output from the ultrasound transducer during pulsed Doppler examination at the settings most commonly used is 71 mW/cm2, which exceeds the limit of 17 mW/cm2 set

by the Food and Drug Administration for ophthalmic examinations but is within the 100 mW/cm2 guidelines suggested by the American Institute for Ultrasound in Medicine. For this reason, the amount of time spent obtaining pulsed Doppler recordings should be minimized. If color Doppler imaging is necessary, it should be performed with minimal pressure on the globe and for the shortest possible time [8].

Gray-Scale US Anatomy: The normal anatomy of the orbit as seen on gray-scale US. The lens, iris, and ciliary body can be seen routinely within the otherwise anechoic globe. The optic nerve-optic nerve sheath complex appears as a hypo echoic stripe posterior to the globe that extends to the orbital apex. The retrobulbar fat is hyper echoic, and the extra ocular muscles are slightly hypo echoic bundles that course from the globe to the orbital apex. The lacrimal gland can often be identified in the superolateral orbit.

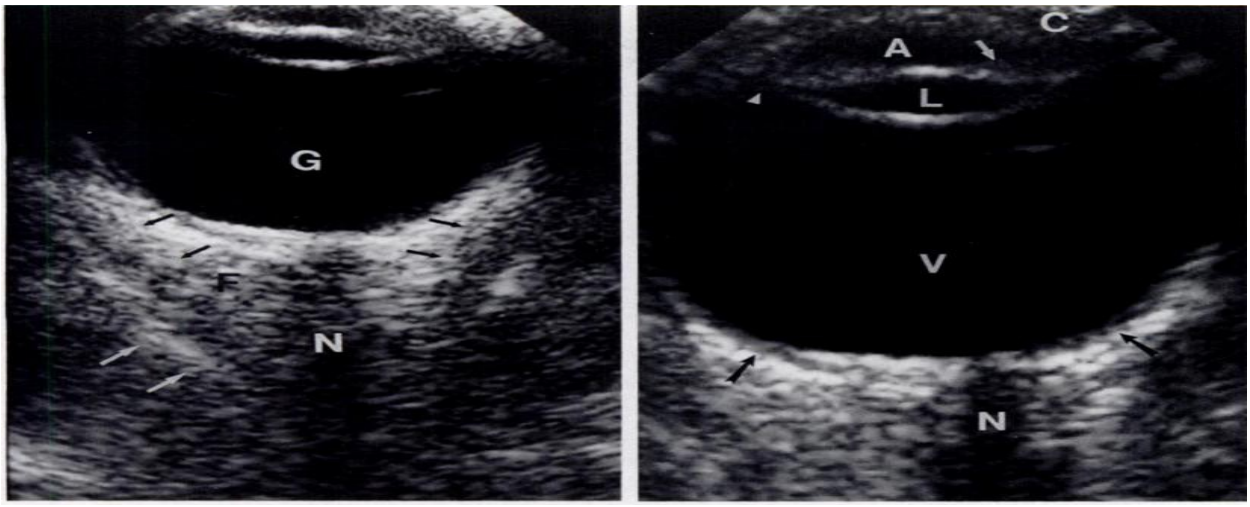


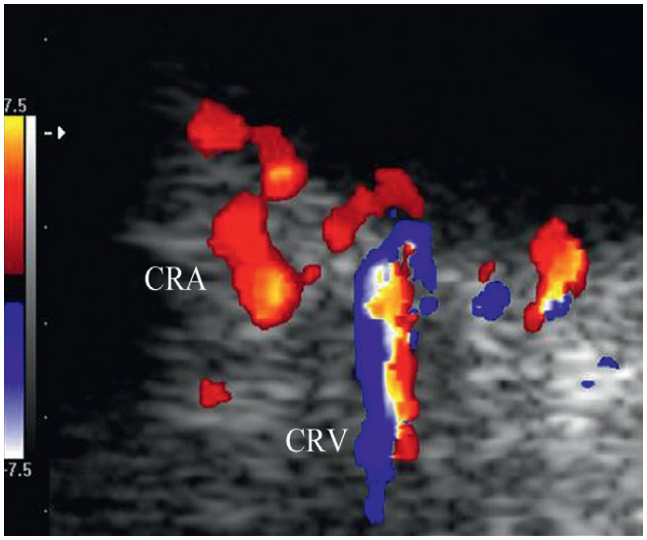
Fig.3.14: Normal gray-scale US anatomy. (a) Transverse gray-scale US image through the orbit demonstrates the anechoic globe (G) anteriorly. The hypo echoic optic nerve-optic nerve sheath complex (N) extends posteriorly from the globe. Retrobulbar fat (F), extra ocular muscles (small arrows), and the orbital wall (large arrows) are visible. (B) Transverse gray-scale US image through the globe demonstrates the vitreous body (V), retina and sclera (black arrows), optic nerve-optic nerve sheath complex (N), lens (L), anterior chamber (A), ciliary body (arrowhead), iris (white arrow), and cornea (C).

Table.3.4: Normal blood flow velocity (cm/sec) in orbital vessels.

Vessel	Doppler signal topography	Peak systolic velocity (mean ±SD)	Peak end diastolic velocity (mean ±SD)
Ophthalmic artery (OA)	Posterior orbit: Lateral to optic nerve	45.3 ± 10.5	11.8 ± 4.3
Central retinal artery (CRA)	ON central portion	17.3 ± 2.6	6.2 ± 2.7
Posterior ciliary arteries (PCA)	Fat posterior to the globe	13.3 ± 3.5	6.4 ± 1.5
Superior ophthalmic vein (SOV)	Superior and nasal orbit	−7.6 ± 1.8	–
Central retinal vein (CRV)	ON central portion	−4.2 ± 0.8	–
Vortex vein	Oblique positions of the posterior globe	−8.5 ± 2.2	–

SD: Standard deviation; ON: Optic nerve
Modified from: Tranquart F, Berges O, Koskas P, et al. Color Doppler imaging of orbital vessels: personal experience and literature review. J Clin Ultrasound 2003;31:258–273.

Fig.3.15: Color Doppler imaging parameters in normal subjects. CDI of the orbit using 5–12 MHz linear transducer showing CRA & CRV



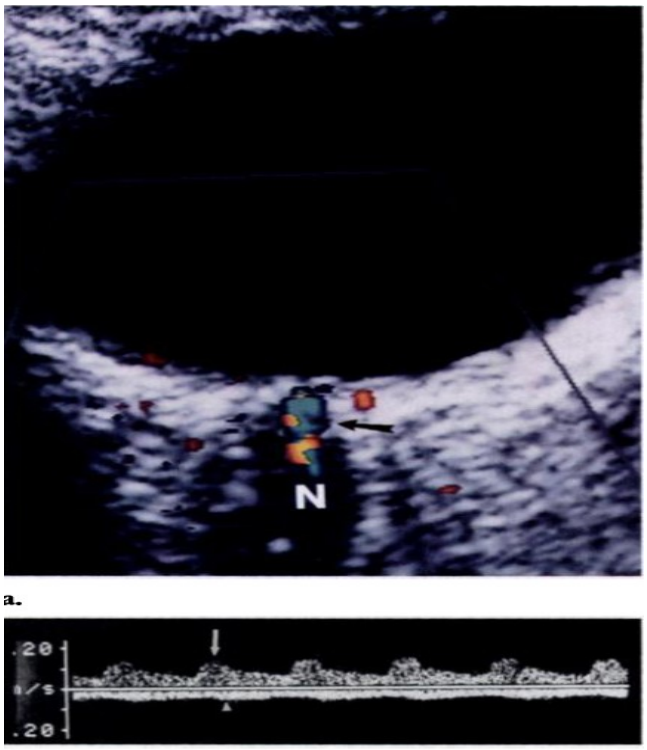


Fig.3.16:CRA and CRV. (a) Transverse color flow image demonstrates the normal CRA and CRV (arrow) in the center of the distal optic nerve (N).
(b) Pulsed Doppler recording of typical waveforms from the central retinal vessels shows the arterial waveform above the zero axis and the venous waveform below the zero axis. Note the rounded systolic peak (arrow) and the continuous flow during diastole in the arterial waveform. The venous flow is low and continuous, with a peak in flow velocity (arrowhead) a fixed time after the systolic peak.

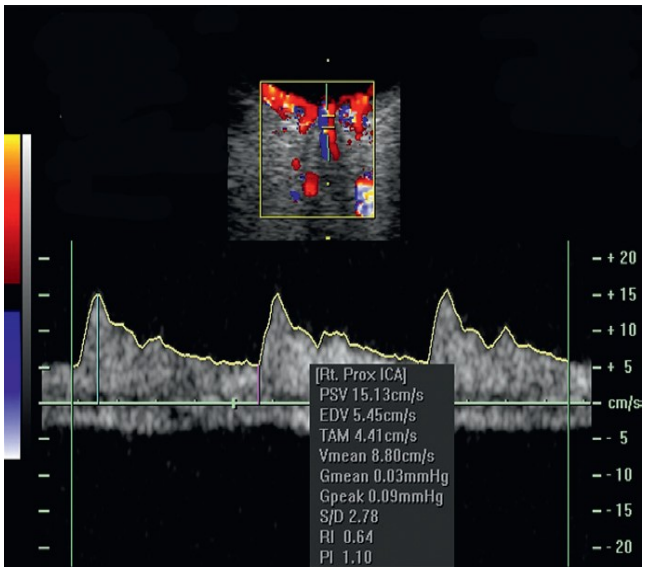


Fig.3.17: Spectral Doppler analysis in normal subjects. Pulse waveform shows pulsatile arterial component above the horizontal line and laminar venous flow below the horizontal line. Peak systolic velocity (PSV) of the CRA: 15.13 cm/s. Resistance index (RI) of the CRA: 0.64 (A)

The waveform of the CRA is characteristic of a low-resistance system. It consists of a rounded systolic peak followed by a slow decline in velocity, with flow continuing throughout diastole. The waveform of the CRV is obtained in the same spectrum as that of the CRA. It consists of relatively low, continuous flow in the opposite direction relative to arterial flow.

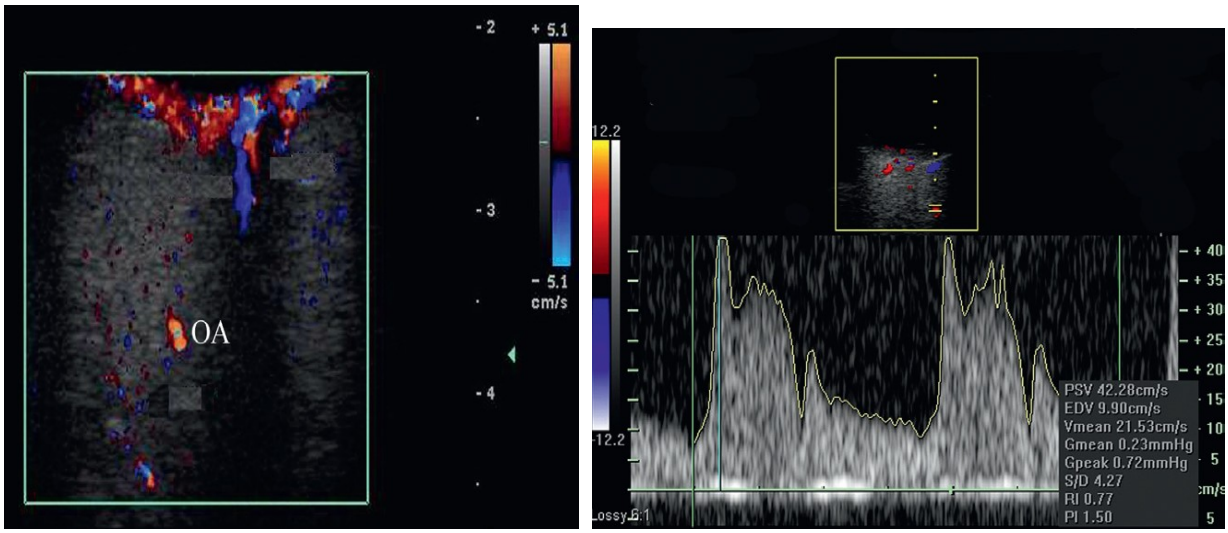


Fig.3.18: Ophthalmic artery .A systolic peak, dicrotic incisura, slightly diastolic flow declination, PSV: 42.28 cm/s, and RI: 0.77 are observed

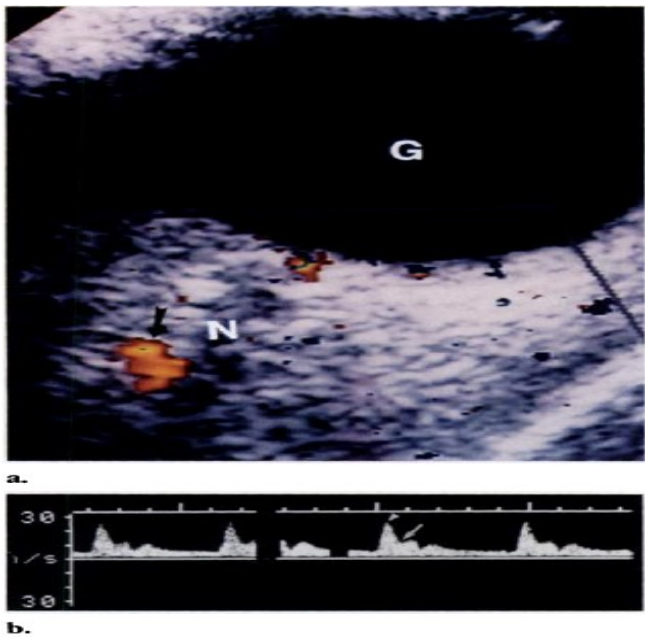


Fig.3.19:OA. (a) Transverse color flow image demonstrates the normal location of the OA (arrow) medial to the optic nerve (N) in the midorbit. G =globe. (b) Pulsed Doppler recording of the normal OA waveform shows a sharp systolic peak (arrowhead),a dicrotic notch (arrow), and relatively little flow in diastole. On all pulsed Doppler images, scale at side of image is in meters per second and scale at top of image is in seconds.

The waveform of the OA is typical for a relatively high-resistance artery. There is a sharp initial peak followed by an incisura and relatively little flow during diastole..

Previous similar studies:

The literature says the existence of hemodynamic alterations in the vessels of the eyeball observed in patients with Diabetes mellitus .However, some authors report different results. This may be a due to assessment of vascular changes in various stages of diabetes mellitus and different criteria of patient selection. This heterogeneity concerns the types of diabetes mellitus and the duration, the status of diabetic retinopathy, co morbid conditions, associated ocular lesions and Doppler parameters. The main component in these observations is the assessment of Doppler flow parameters and their significance in the prognosis of diabetic retinopathy and its monitoring.

Goebel *et al* [44] were the first ones to show a high sensitivity of this method in monitoring hemodynamic changes in patients with diabetic retinopathy. They compared patients with simple, non proliferative and proliferative retinopathy. They found a significant decrease in PSV and EDV in the CRA as compared to healthy normal controls. A decrease in blood flow velocity in the retrobulbar vessels was observed in all groups, which correlated with the progression of diabetic retinopathy. No significant difference in Doppler flow parameters in the OA and PCAs were observed. They did not calculate the resistivity index.

Kawagishi *et al*.[45] found the decrease in PSV and EDV in the CRA. They found the changes in the CRA before clinical features of Diabetic retinopathy appeared. The RI was found to be elevated and correlated with the blood glucose level.

Güven *et al*.[46] reached the same results wherein they noted reduction in PSV in the CRA in patients with NPDR and PDR as well as in patients following retinal laser pan photocoagulation. Here, the RI index was not calculated either and there was no significant differences in the flow velocities of OA and PCAs.

Mendivil *et al*.[47] noticed a significant reduction in PSV and EDV in the OA and CRA in patients with proliferative retinopathy compared to healthy normal individuals. No velocity disturbances were noted in the PCAs. They [48] also studied the influence of laser pan photocoagulation on blood flow velocity and found that retinal photocoagulation caused a significant reduction in the flow velocity of OA, CRA and CRV. These parameters did not change in a significant manner in a period of one year of follow-up.

Arai *et al*.[49] emphasized the significance of the RI index in the CRA and OA. According to them, it is a more sensitive Doppler parameter for predicting the severity of diabetic retinopathy than Doppler flow velocity measurements in these vessels. Greater vascular resistance may be seen prior to retinopathy. They claimed that measurements in the CRA are more significant for hemodynamic alteration assessment in diabetes than measurements in the OA. The PSV and EDV values in the CRA were significantly lower in both diabetics with retinopathy and without retinopathy. The differences noticed were dependent on the severity of retinopathy. CRA is more sensitive to auto regulation disorders which show changes even before any findings noted in the fundus. PSV in the OA showed no differences between the groups.

Evans *et al*[50] did their studies in hyperoxic conditions in normal healthy individuals and in diabetic patients without or with minimal retinopathy changes in the ocular fundus. Both study groups were examined during regular normal breathing and after oxygen saturation. In hyperoxic conditions, the EDV and RI changed in a significant manner in healthy individuals as EDV decreased and RI increased. In contrast, the diabetic patients examined under hyperoxic conditions showed increased EDV in the CRA and decreased RI compared to the normal control group. No statistically significant changes in the OA found in the healthy controls.

Ino-ue *et al*.[51] studied changes in blood flow velocities in the OA in patients with ocular ischemic syndrome and with simple as well as proliferative diabetic retinopathy. The persons with diabetic retinopathy presented lower EDV and higher PI values when compared with the control group, and persons with ocular ischemic syndrome and diabetes showed significantly reduced systolic and diastolic velocities and increased pulsatility indices. There were no significant differences between the groups and no relation was noticed between the flow velocity of orbital vessels and progression of diabetic retinopathy.

MacKinnon *et al*.[52] studied flow velocities in the OA and CRA as well as the RI index in three groups of patients: without diabetic retinopathy or with simple retinopathy, with preproliferative or proliferative lesions and in healthy normal controls. In the group of patients with preproliferative and proliferative retinopathy, PSV and EDV in the CRA were significantly reduced than in patients without lesions or with simple retinopathy. In

comparison with the control group, the RI index was considerably increased in the OA and decreased in the CRA. In the OA, however, no difference in velocities was noted between the examined groups.

Dimitrova *et al*[53]in their study compared the results of blood flow measurements in patients with simple retinopathy following 21 months from the primary examination and assessed the influence of the disease progression on hemodynamic changes. After this period of time, they observed no changes in the flow parameters were in the CRA and ciliary arteries. The authors suggest that, with the progression of diabetic retinopathy, initial lesions develop in the central retinal vein.

Krepler *et al*.[54]studied the effect of vitrectomy done in patients with diabetic retinopathy on the ocular blood flow. The mean flow velocity in the CRA and PCAs decreased in a significant manner, and the RI value reduced in the CRA with no change in the PCAs after vitrectomy. The results showed that the vitrectomy may cause a reduction in the ocular blood flow.

Sullu *et al* [55] performed similar studies and assessed orbital blood flow parameters in the first and sixth months after the vitrectomy procedure. A significant decrease of the Resistivity index in the CRA was found after the procedure.

Gracner [56] observed the Doppler parameters in the OA, CRA and PCAs on various stages of diabetic retinopathy development and showed a significant increase in PSV in the Opthalmic artery with advanced preproliferative and proliferative lesions as well as a decrease in PSV and EDV in the Central retinal artery when compared to the control

group. They also noted a reduction in EDV in the PCAs. The RI index in the Opthalmic Artery and Central retinal artery did not change in a significant manner, but increased in the PCAs. The author suggested that the results may be useful in assessing the progression of the disease.

Kraśnicki *et al*.[57] assessed blood flow velocities in the orbital vessels in patients with type II diabetes in a group without lesions in the ocular fundus and in another group with the presence of non-proliferative retinopathy lesions. They found significantly lower PSV and EDV values in the OA in both groups compared to healthy controls. The RI did not alter in a statistically significant manner. In the CRA, PSV and EDV were significantly lower only in the group with more severe diabetic retinopathy lesions. The RI was significantly higher in patients with more advanced retinopathy lesions. In the PCAs, the significant difference was observed only in the PSV in the group of patients with non-proliferative diabetic retinopathy group. The authors stressed the role of Colour Doppler ultrasonography and the influence of decreased blood flow parameters in the CRA and ciliary arteries on the development of retinopathy.

Yilmaz Ovali *et al*.[58] assessed the orbital vessels in pediatric patients with type I diabetes but without retinopathy and detected alterations in the blood flow parameters in the OA and CRA when compared with healthy peers. In patients with diabetes mellitus for more than 5 years, EDV in the OA was significantly higher and the RI was lower. The RI in the CRA increased in patients with higher levels of microalbuminuria. The

examination revealed early lesions in the OA that were not as significant as reported by other studies [45, 49, 52].

Modrzejewska *et al*[59] assessed blood flow parameters of the vessels in the eyeball and their relation to the blood lipid values in the group of young patients with type I diabetes who had initial vascular lesions in the fundus. The study showed a decrease in blood flow parameters when the vascular lesions in the ocular fundus were at their initial stage. Peak Systolic Velocity, Mean Velocity (MV) and Resistivity Index in the CRA as well as PSV, MV and PI in the temporal posterior ciliary arteries (TPCAs) values were found to be decreased. The differences in total cholesterol level, HDL, LDL cholesterol and apolipoproteins (ApoB) were associated with blood flow resistance alterations, mainly in the OA. They suggested that the effect of lipid disorders on the risk of retinal ischemia in young patients with type I diabetes mellitus may be significant.

Dimitrova *et al.* [60] studied the relation between diabetic retinopathy and arterial hypertension on the basis of flow analyses in the central retinal vein and artery in four groups of patients. PSV and EDV velocities were the highest in the group of patients without retinopathy and hypertension, as well as in patients without retinopathy but with systemic hypertension. The Pourcelot index in the CRV was significantly lower in both groups. A significant relation was noted between systemic hypertension, status of diabetic retinopathy and PSV in the CRA.

Lockhart *et al.*[61]used novel techniques of spectral Doppler analysis for a quantitative assessment of blood flow changes in the orbital vessels and carotid arteries .The aim of

the study was to study whether it is possible to detect subclinical circulation disorders in patients with uncomplicated Type I diabetes mellitus. The authors identified measurable differences in blood flow velocities much earlier to the development of profound retinopathy.

Pemp *et al*.[62] studied the relation between blood flow parameters in the CRA measured in a color Doppler examination and blood flow parameters in the CRA assessed with a laser Doppler flow meter with regard to the caliber of the vessel. The groups included patients with type I DM with mild retinopathy and without it, and they were compared with normal individuals. The patients with diabetes showed greater vascular diameter of the CRA, but no significant blood flow parameter differences were noticed. The authors recommended caution in interpreting the results as the use of color Doppler sonography does not provide information on the vascular diameters.

4.AIM OF THE STUDY

The Aim of this study is to compare orbital vessel Doppler indices in diabetics with retinopathy and diabetics/healthy controls without retinopathy using the color Doppler sonography. The Peak Systolic Velocity, End Diastolic Velocity, Pulsatility Index and Resistivity Index are measured in Opthalmic artery, Central retinal artery and Central retinal vein in the three groups and compared.

5.METHODOLOGY

STUDY CENTRE: Barnard Institute of Radiology,

Madras Medical College,

Rajiv Gandhi Govt General Hospital, Chennai

STUDY PERIOD: JUNE 2017 – MAY 2018

NUMBER OF HEALTHY CONROLS: 90

NUMBER OF DIABETIC PATIENTS WITHOUT RETINOPATHY: 68

NUMBER OF PATIENTS WITH RETINOPATHY: 55

DURATION OF STUDY: ONE YEAR

STUDY DESIGN: Prospective study

INCLUSION CRITERIA :

- Age between 20 and 80 years, both sexes.
- Cases with non proliferative diabetic retinopathy.
- Diabetics without retinopathy
- Non-diabetic healthy controls were included in the study.

EXCLUSION CRITERIA: Exclusion criteria were

- Previous laser photocoagulation
- Proliferative diabetic retinopathy
- Any disease or anomaly of the eye, which may affect blood flow velocity, such as ocular inflammation, systemic diseases like hypertension, Non-diabetic vascular disease.
- Trauma
- Lactating and pregnant females whatever the gestational age.

METHODOLOGY:

This prospective study was performed after obtaining clearance from our Institutional Ethics Committee and institutional informed consent guidelines were observed. The patients were screened using the drawn inclusion/ exclusion criteria. Relevant entries in the proforma for each patient were made after reviewing his/her case sheet & previous medical records.

STUDY POPULATION:

The study population included diabetic patients without retinopathy, with retinopathy, non diabetic healthy controls who came to diabetology outpatient department after fasting and post prandial blood investigation. Fundoscopy was performed by expert ophthalmologist .Patients with retinopathy and no retinopathy was categorized based on International disease severity scale for DR by Wilkinson which proposes five levels for grading of DR, based on risk of progression Classification is worded as follows: none, mild non proliferative diabetic retinopathy, moderate non proliferative diabetic retinopathy, severe non proliferative diabetic retinopathy or proliferative diabetic retinopathy. Normal healthy controls were also taken from general medicine Out Patient Department who comes for diabetic screening purpose. Systolic and diastolic blood pressure measurements were done.

The final population enrolled in this study composed of 55 patients with non proliferative diabetic retinopathy, 68 diabetic patients without retinopathy, 90 healthy controls without

diabetic retinopathy. All patients were required to provide written informed consent before study participation.

COLOUR DOPPLER IMAGING: Patients to be in supine position and sterile gel will be placed in closed eyelid. Measurements include Peak Systolic Velocity (PSV), End Diastolic Velocity (EDV) in ophthalmic arteries, central retinal artery and central retinal vein. Resistive Index (RI) and Pulsatile Index (PI) will be measured by the following formula.

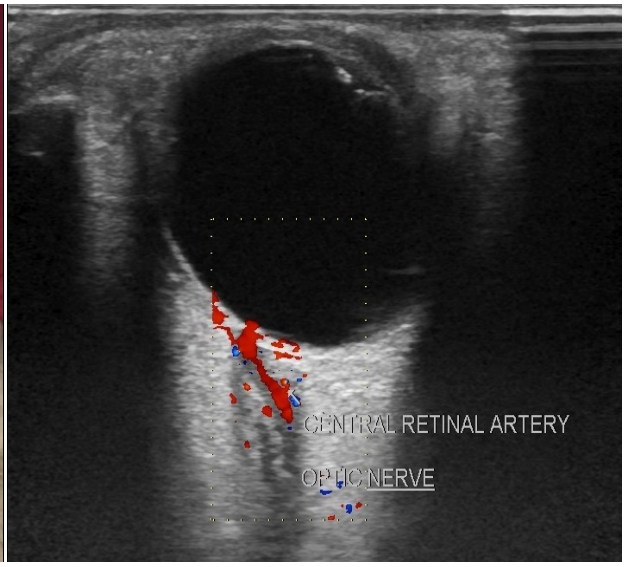
Resistive Index (RI) = PSV-EDV/PSV; Pulsatile Index (PI) = PSV-EDV/V mean;

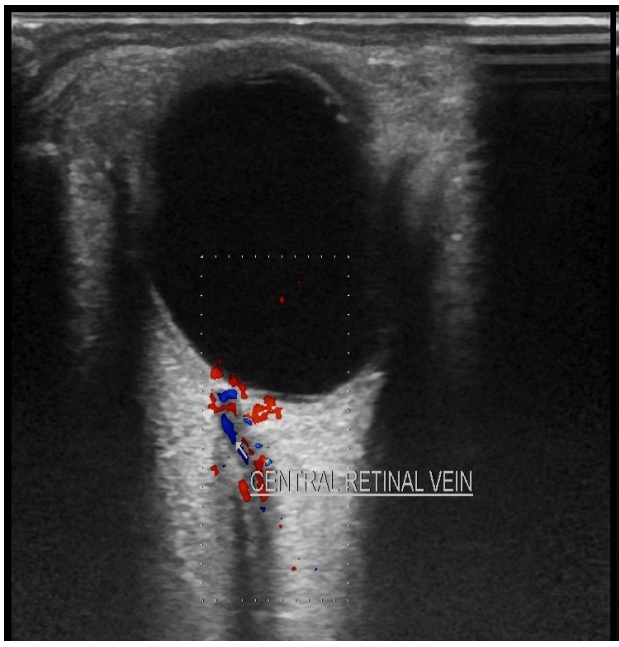
V mean=1/3(PSV-EDV) + EDV.

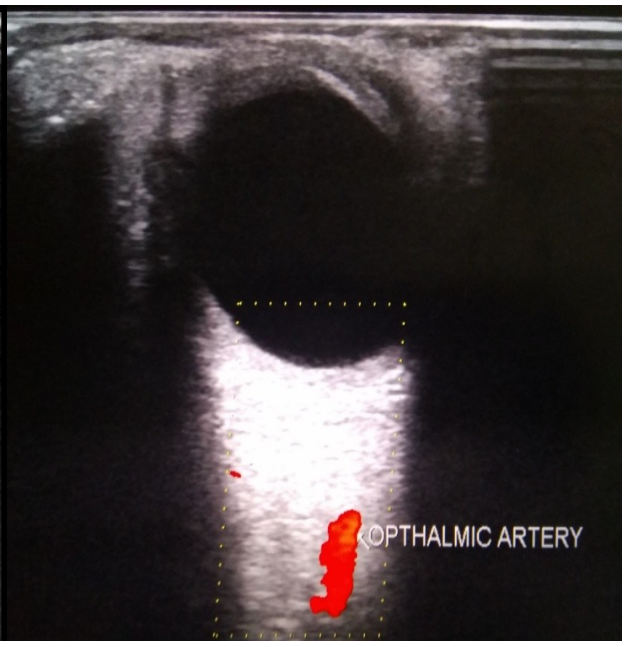
The eye with severe retinopathy is selected and if retinopathy is equal in both eyes, the right eye is selected for further evaluation. The signal localized from ophthalmic artery in upper part of intraocular region to optic nerve. The RI of OA was measured near the nasal side of the optic nerve where it crosses OA, behind the 3–5 cm of globe. CRA was visualized on the localization of the optic nerve head and measurements were taken 5–10 mm behind the optic disc. The ocular vessels identified and flow characteristics i.e., the arterial flow is pulsatile and is usually red (toward the probe) whereas venous flow has continuous spectrum and is blue (away from the probe). CRA waveform shows pulsatile arterial flow with a steep systolic peak suggesting a high-resistance distal vascular bed. The CRV shows a much more continuous waveform but with a small degree of pulsatility. No age specific nomograms were available for PSV and EDV values were available. So wide age range of at risk population was considered. Orbital Doppler indices were compared in each group.

Representative cases

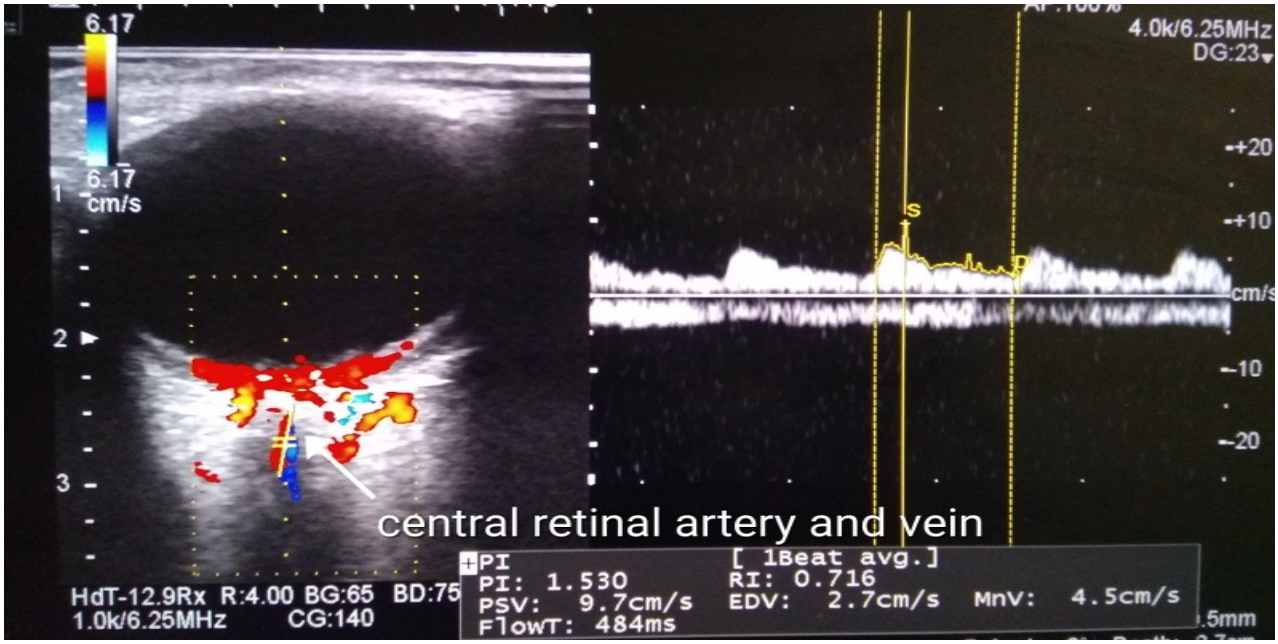
Technique:

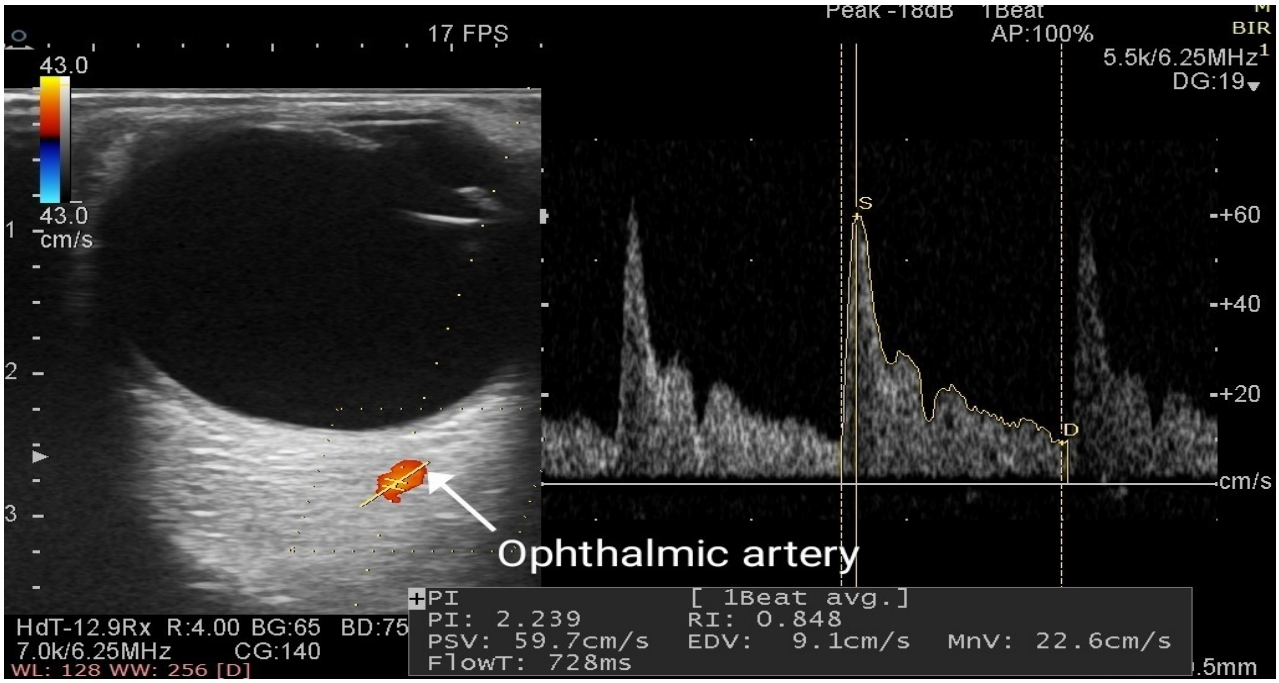




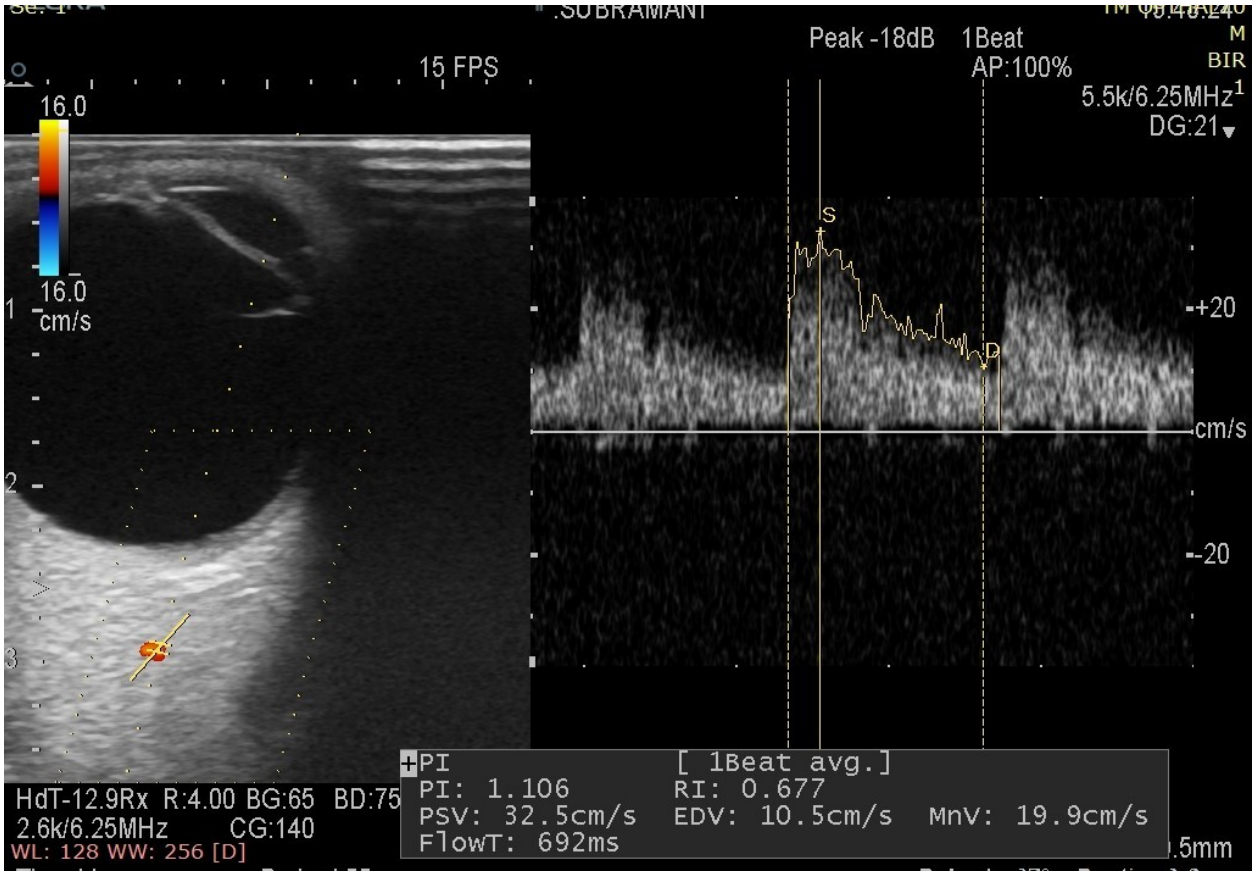


Orbital vessels pulse wave pattern:





CASE:1



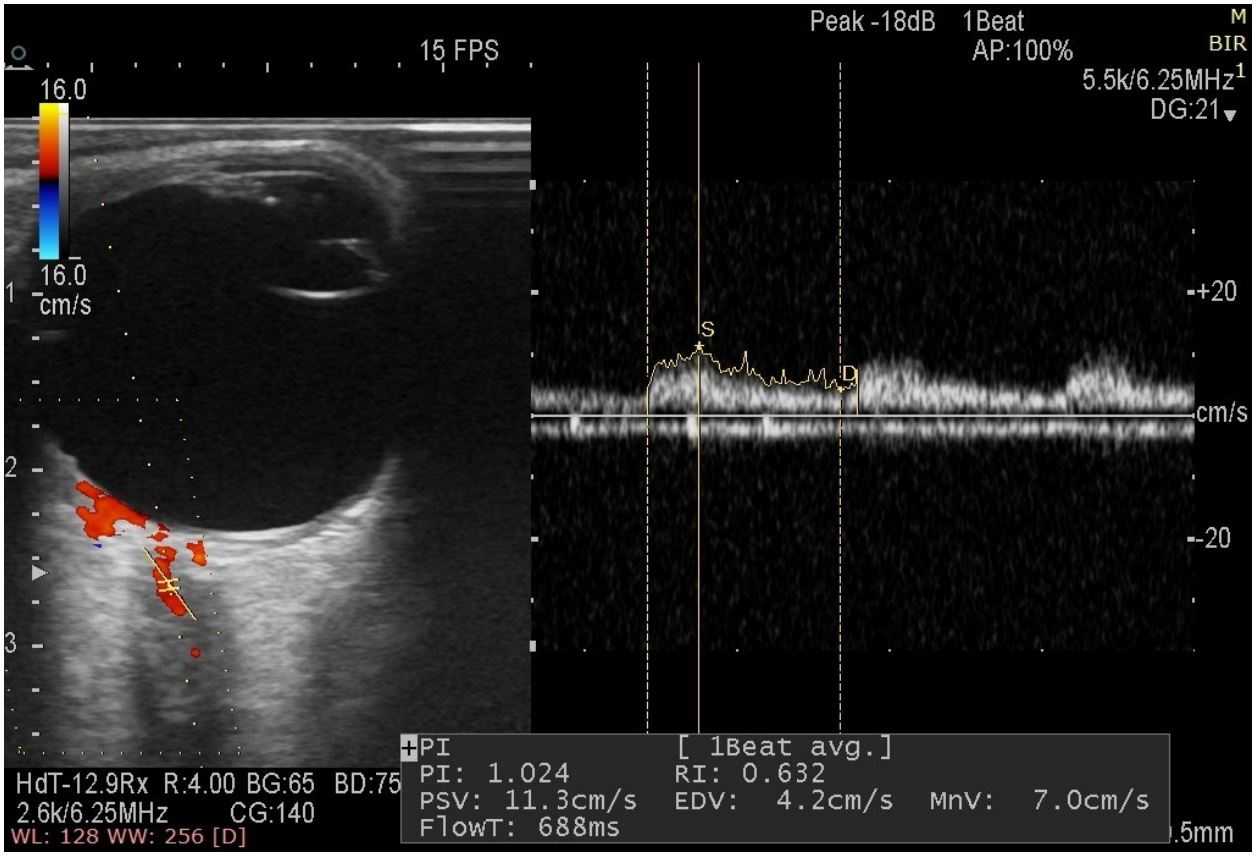
Opthalmic Artery in a normal healthy control showing systolic peak, dicrotic incisura, slightly diastolic flow declination noted.

PSV: 32.5cm/s

EDV: 10.5 cm/s

PI: 1.106

RI: 0.677



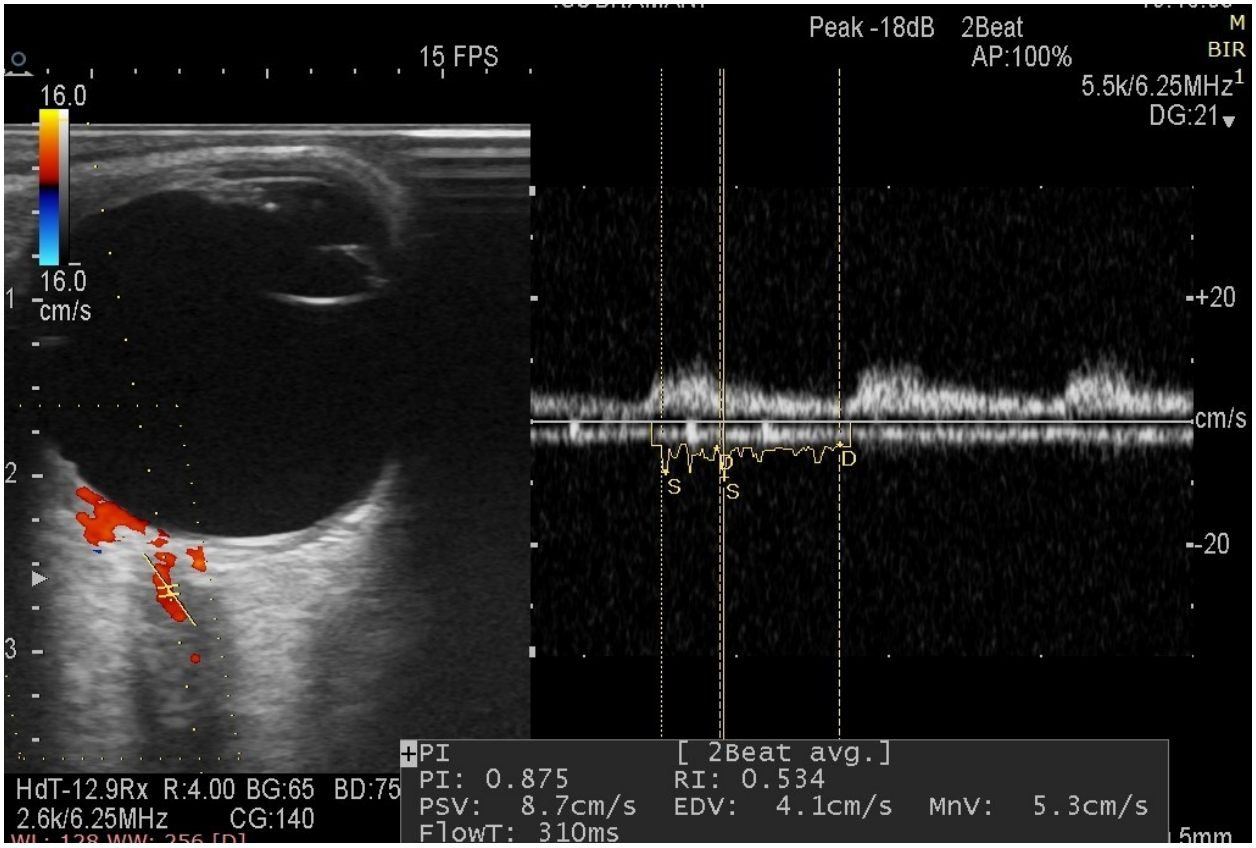
Central retinal Artery in a normal healthy control shows the rounded systolic peak and the continuous flow during diastole in the arterial waveform.

PSV: 11.3cm/s

EDV: 4.2 cm/s

PI: 1.024

RI: 0.632



Central retinal vein in a normal healthy control

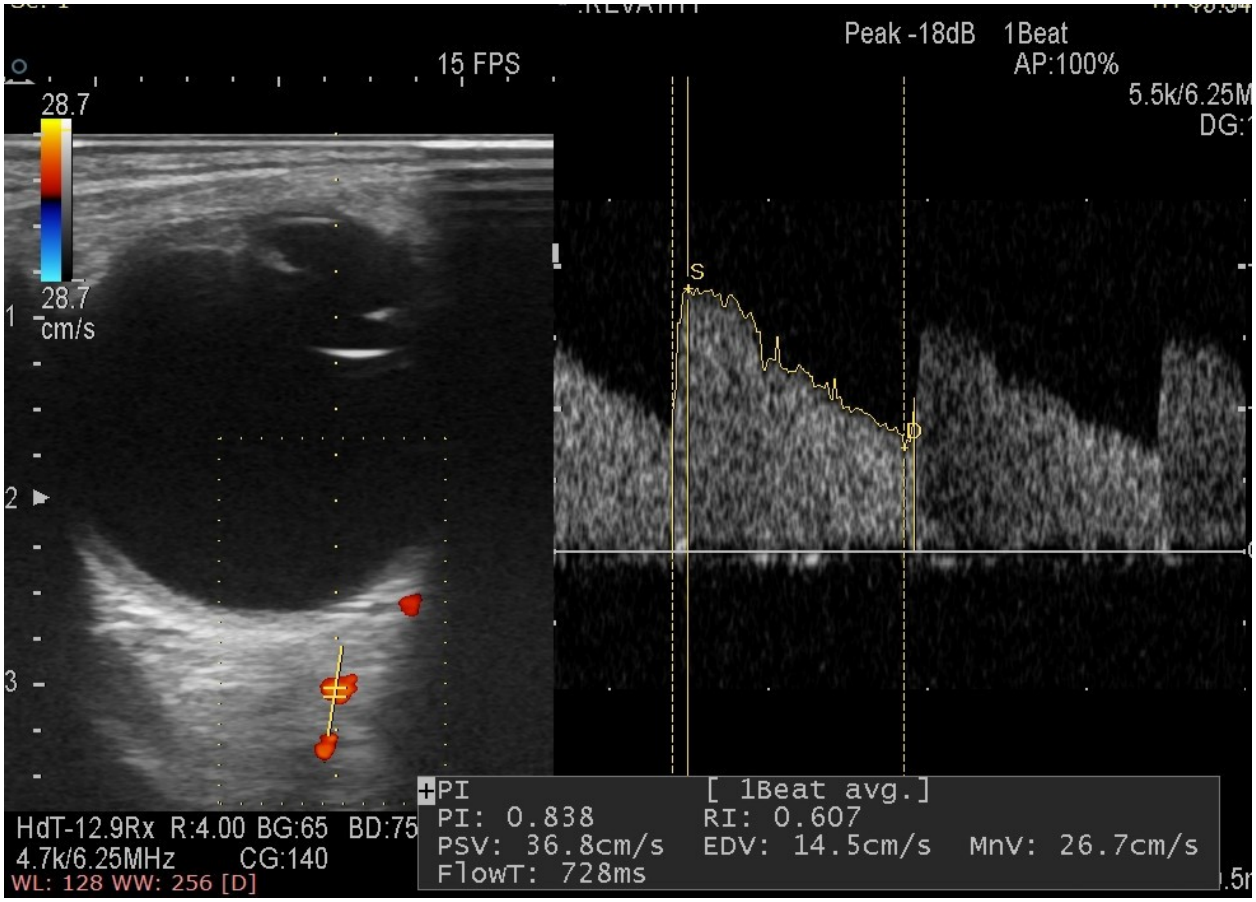
PSV: 8.7cm/s

EDV: 4.1 cm/s

PI: 0.875

RI: 0.534

Case:2



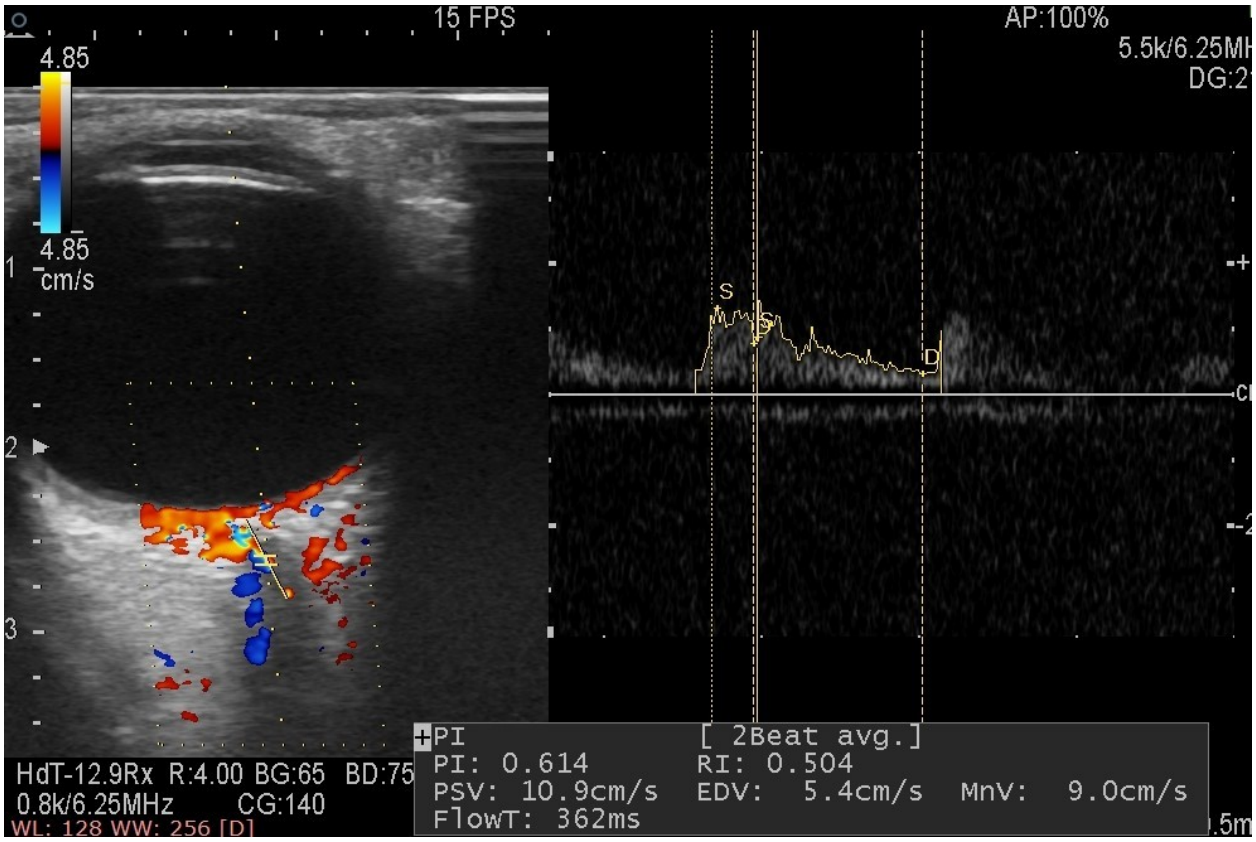
Opthalmic Artery in a normal healthy control

PSV: 36.8 cm/sec

EDV: 4.5 cm/sec

PI: 0.607

EDV: 14.5



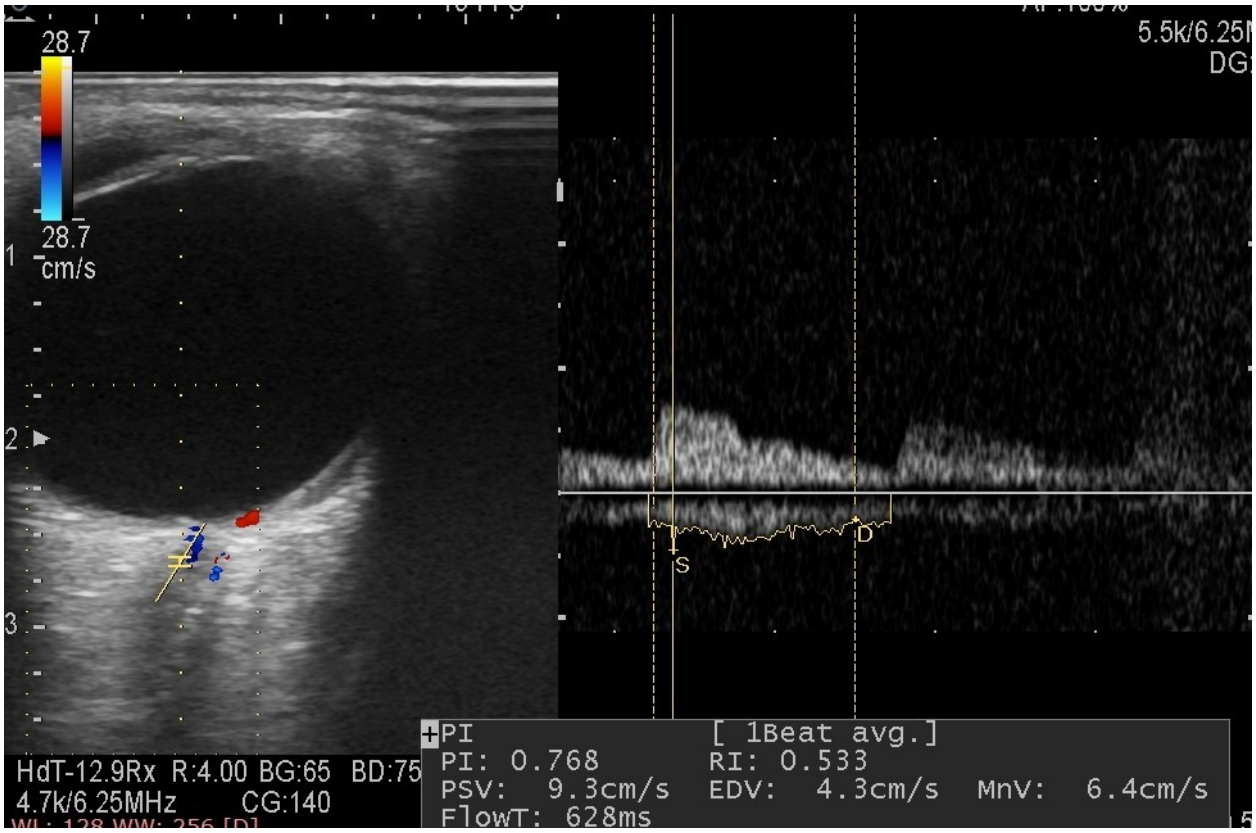
Central retinal artery in a normal healthy control

PSV: 10.9cm/s

EDV: 5.4cm/s

PI: 0.614

RI: 0.504



Central retinal vein in a normal healthy control .Minimal pulsatility noted in the central retinal vein.

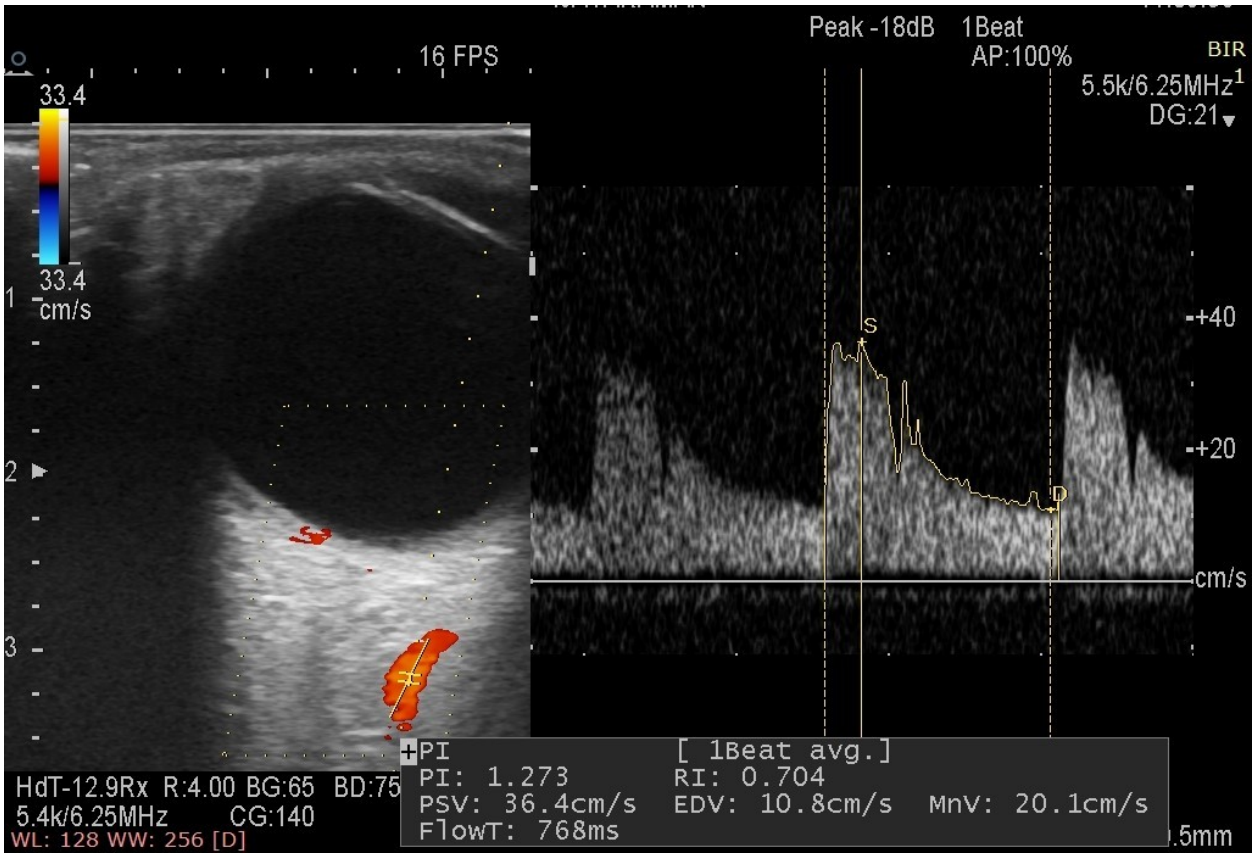
PSV: 9.3cm/s

EDV: 4.3 cm/s

PI: 0.768

RI: 0.533

CASE:3



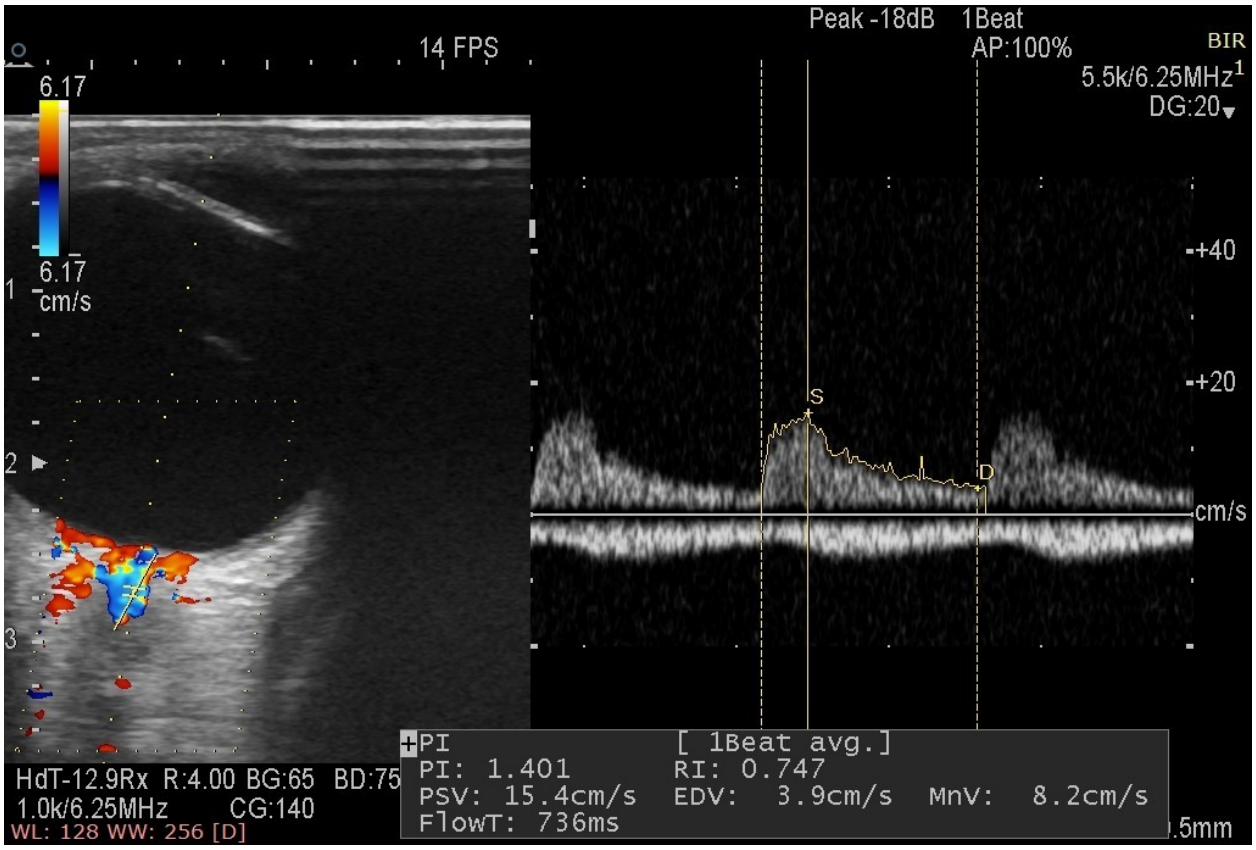
Opthalmic artery in a diabetic without retinopathy

PSV: 36.4 cm/s

EDV: 10.8 cm/s

PI: 1.273

RI: 0.704



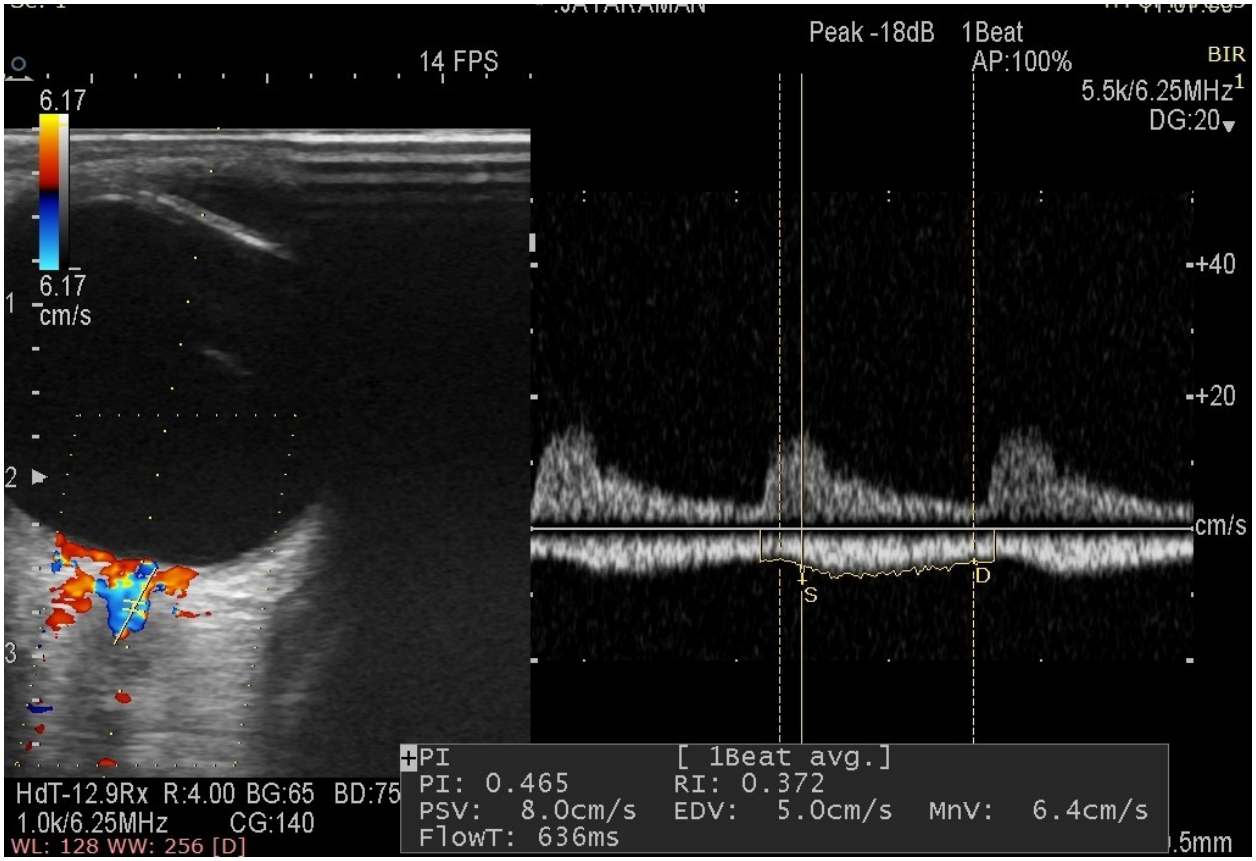
Central retinal artery in a diabetic without retinopathy

PSV: 15.4 cm/sec

EDV: 3.9 cm/sec

PI: 1.401

RI:0.747



Central retinal vein in a diabetic without retinopathy The venous flow is low and continuous, with a peak in flow velocity (arrowhead) a fixed time after the systolic peak.

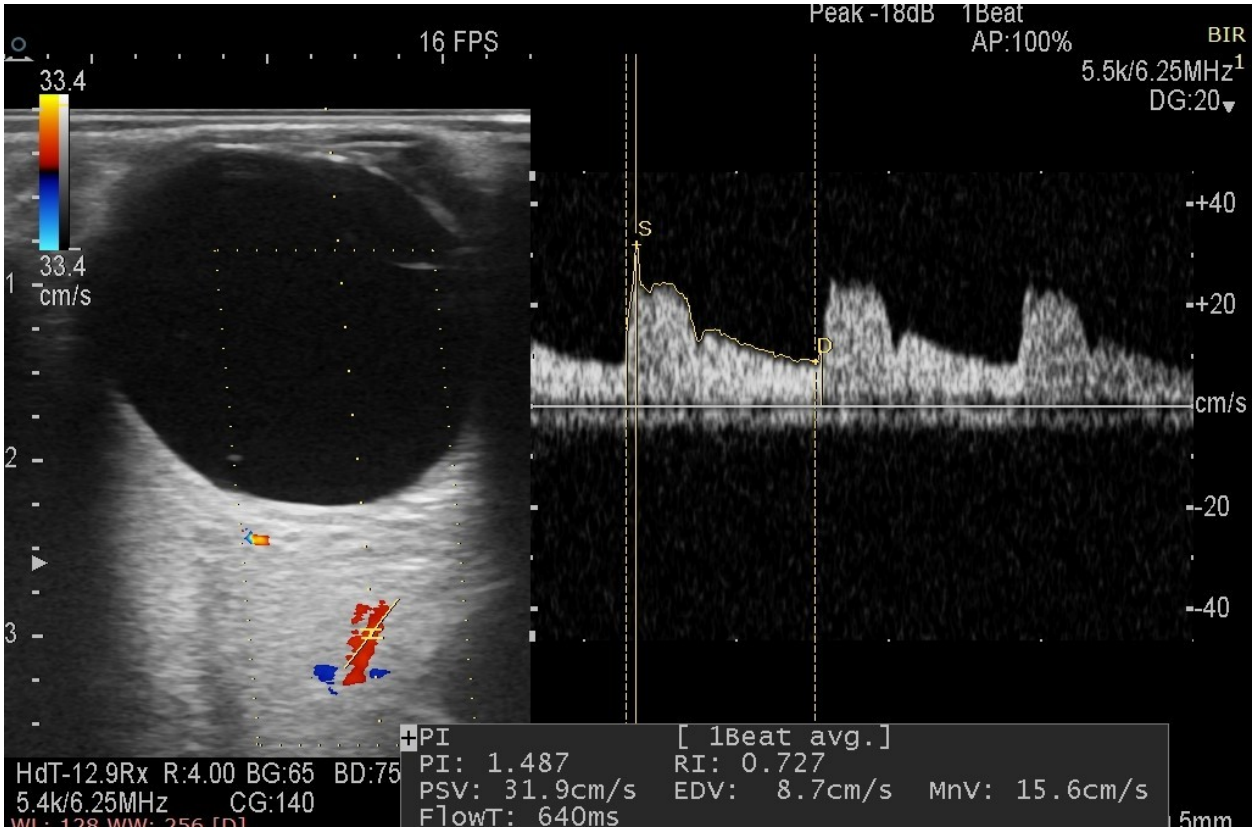
PSV: 8 cm/sec

EDV: 5 cm/sec

PI: 0.465

RI: 0.372

Case:4



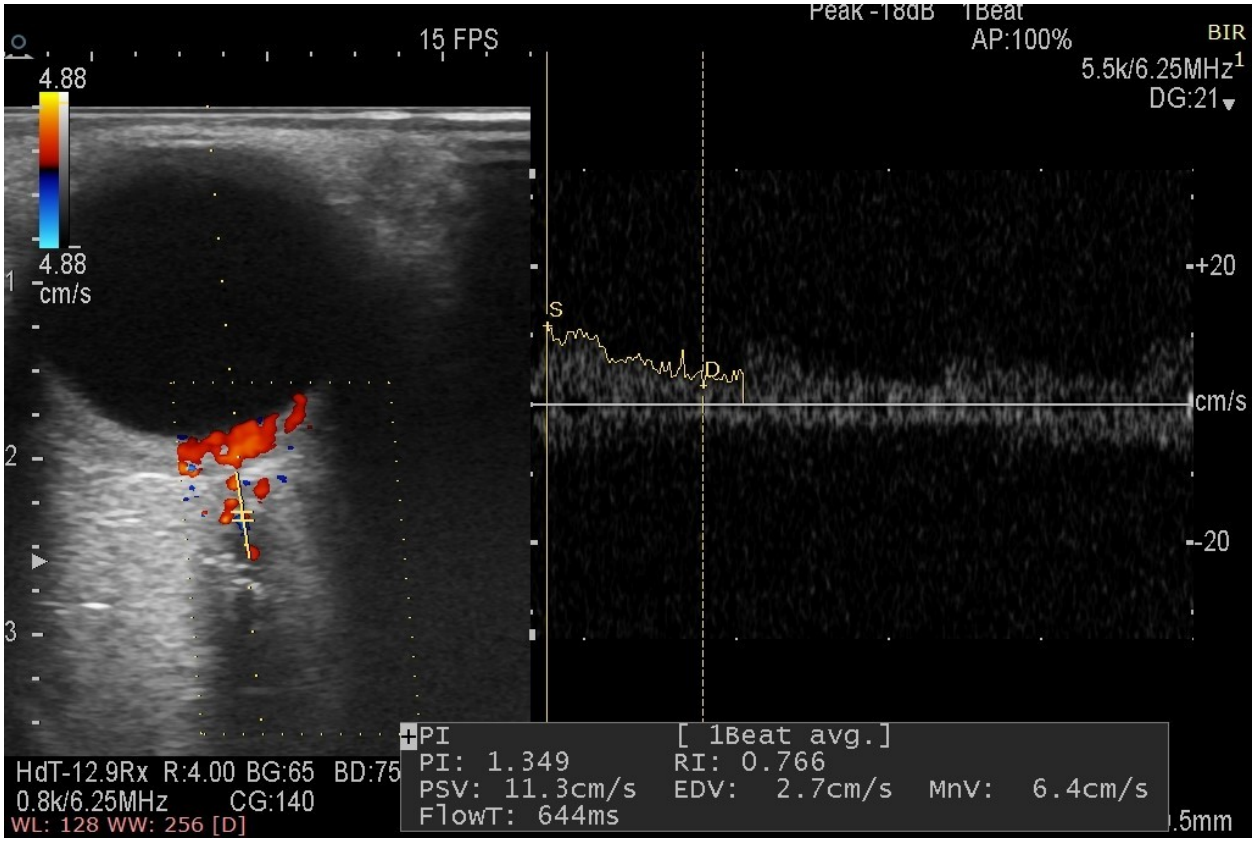
Opthalmic artery in a diabetic patient without retinopathy

PSV: 31.9.9 cm/sec

EDV: 8.7cm/sec

PI: 1.487

RI: 0.727



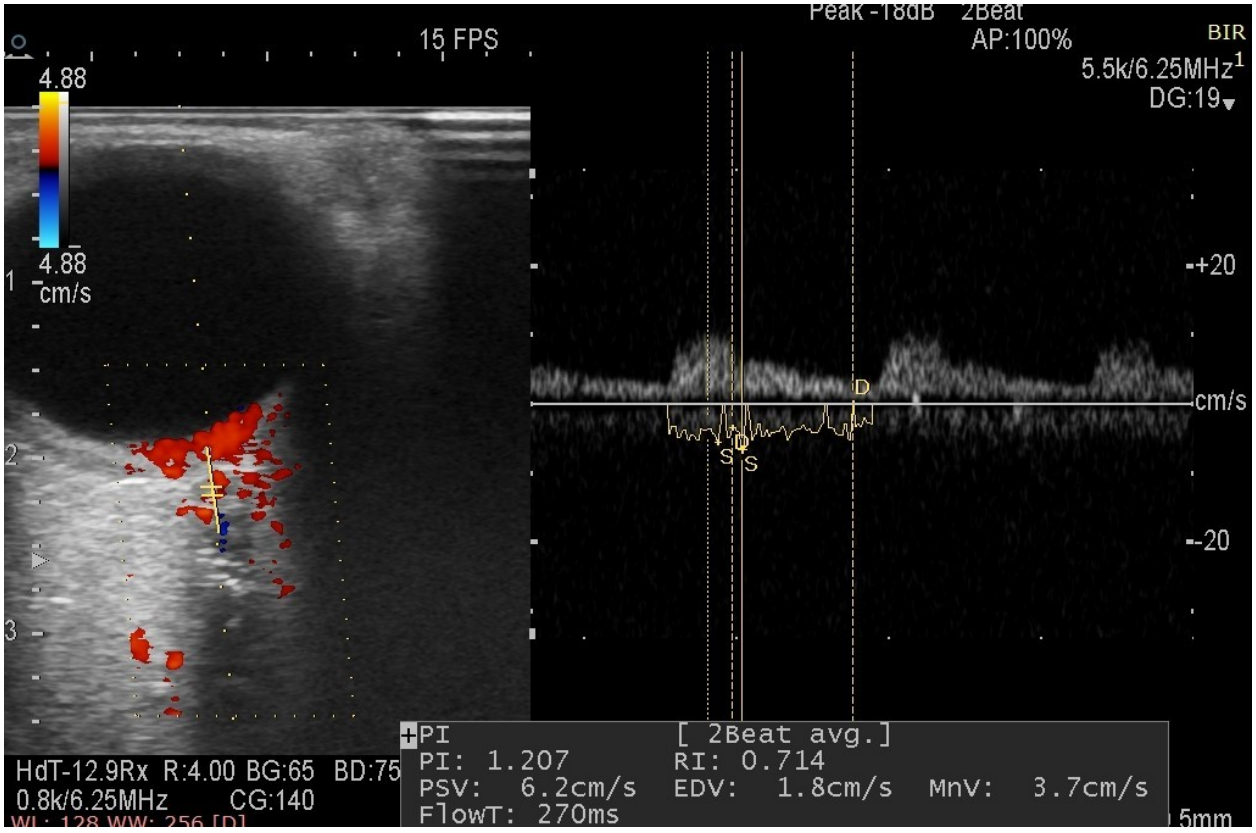
Central retinal artery in a diabetic patient without retinopathy

PSV: 11.3 cm/sec

EDV: 2.7 cm/sec

PI: 1.349

RI: 0.766



Central retinal vein in a diabetic patient without retinopathy

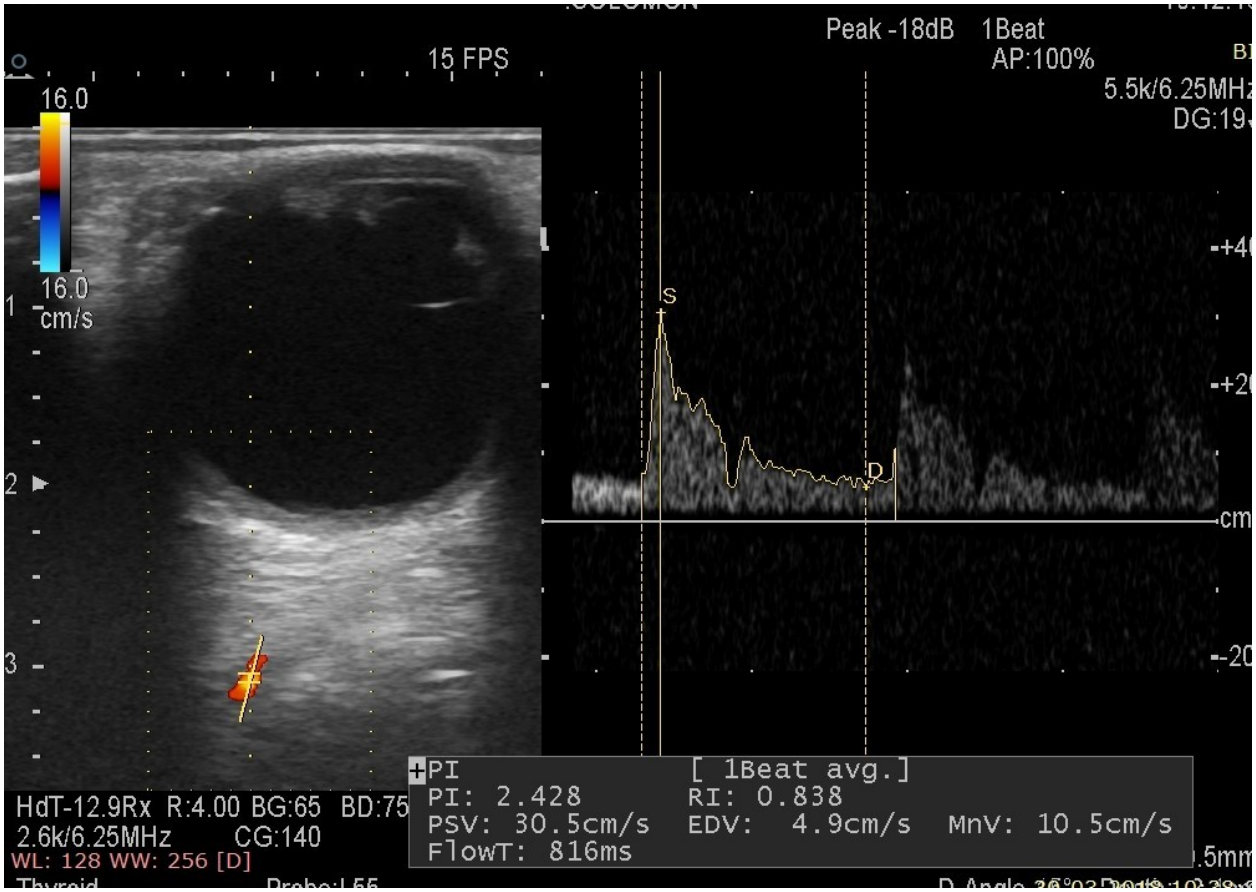
PSV: 6.2cm/sec

EDV: 1.8 cm/sec

PI: 1.207

RI: 0.714

CASE:5



Opthalmic artery in a patient with diabetic retinopathy

PSV: 30.5 cm/sec

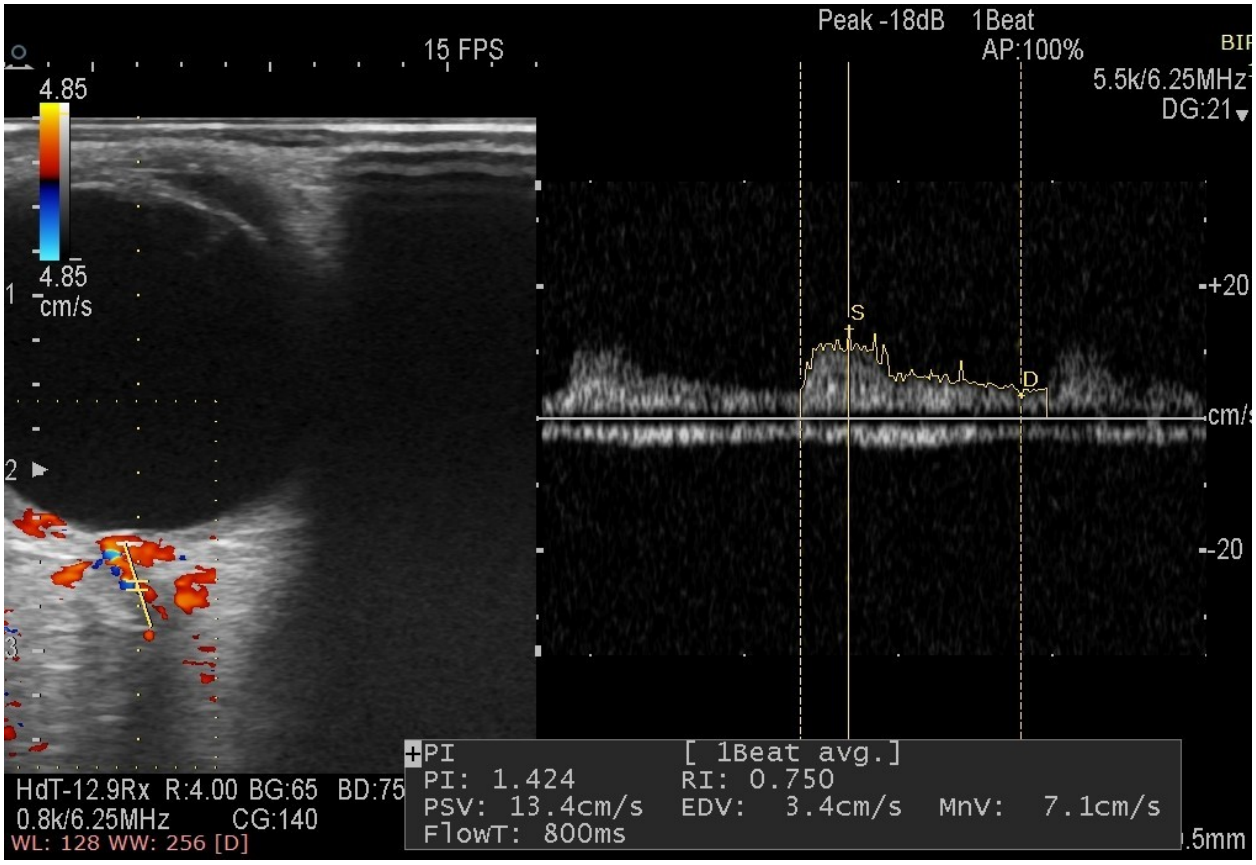
EDV: 4.9 cm/sec

PI: 2.428

RI: 0.838

EDV was found to be lower with increased PI and RI in diabetic retinopathy group.

No statistical significance was found with PSV



Central retinal artery in a diabetic patient with retinopathy

PSV :13.4 cm/sec

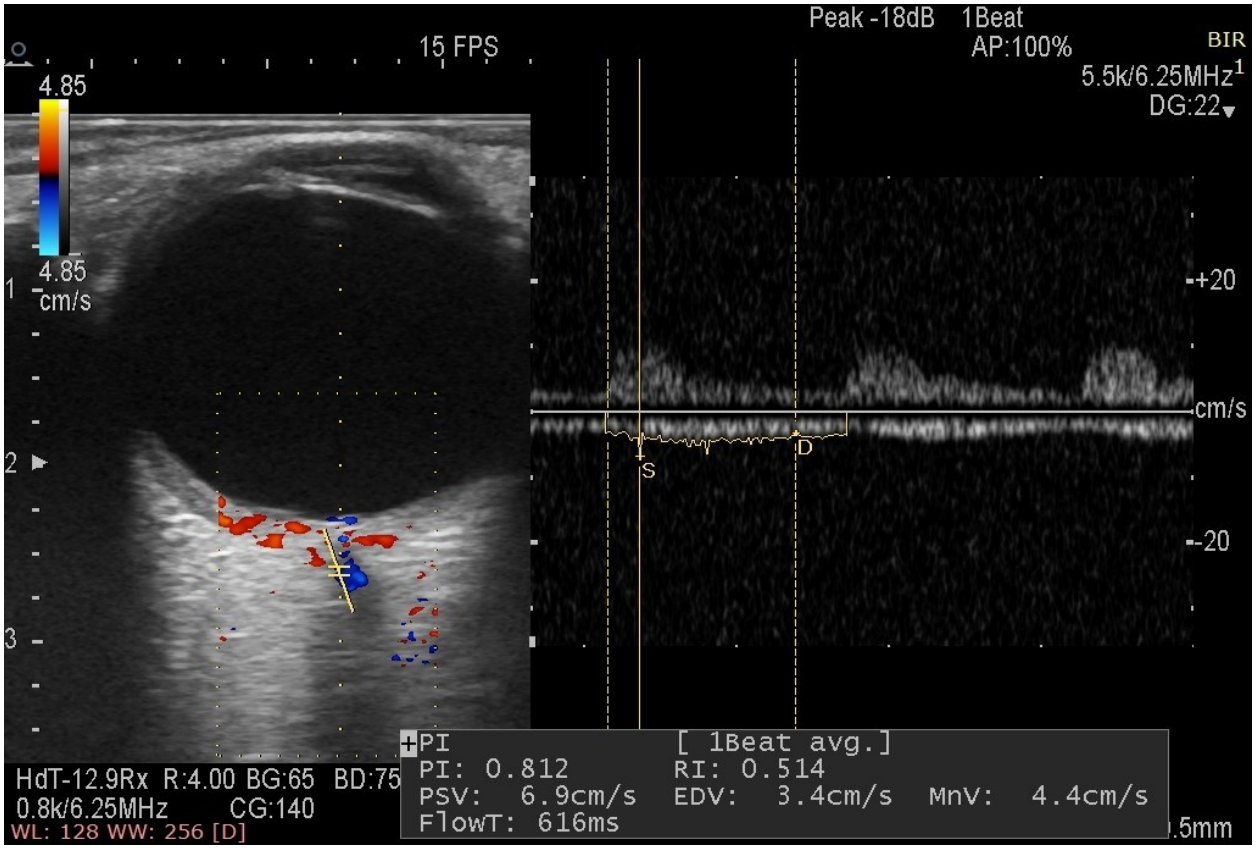
EDV :3.4 cm/sec

PI :1.424

RI :0.750

EDV was found to be lower with increased PI and RI in diabetic retinopathy group.

No statistical significance was found with PSV



Central retinal vein in a diabetic patient with retinopathy

PSV: 6.9 cm/sec

EDV: 3.4 cm/sec

PI: 0.812

RI: 0.514

PSV was found to be higher with increased PI and RI in diabetic retinopathy group

6. STATISTICAL METHODS

Orbital Doppler parameters were considered as primary outcome variable. Study group (Diabetic retinopathy Vs Diabetic without retinopathy Vs Non diabetic) were considered as Primary explanatory variable.

Descriptive analysis was carried out by frequency and proportion for categorical variables. Data was also represented using appropriate diagrams like bar diagram, pie diagram and scatter diagram.

All the age, weight, height, systolic blood pressure, diastolic blood pressure, fasting blood sugar, post prandial blood sugar, HbA1c, ophthalmic artery parameter, central retinal artery and central retinal vein (PSV, EDV, PI, RI) were checked for normal distribution within each category of diabetic retinopathy, diabetic no retinopathy and non-diabetic by using visual inspection of histograms and normality Q-Q plots. Shapiro- wilk test was also conducted to assess normal distribution. Shapiro wilk test p value of >0.05 was considered as normal distribution.

. For non-normally distributed age, weight, height, systolic blood pressure, diastolic blood pressure, fasting blood sugar, post prandial blood sugar, HbA1c, ophthalmic artery parameter, central retinal artery and central retinal vein (PSV, EDV, PI,RI), Medians and Interquartile range (IQR) were compared across study groups(diabetic retinopathy, diabetic without retinopathy and non-diabetic)using Kruskal Wallis test

Categorical gender was compared across study groups (diabetic retinopathy, diabetic without retinopathy and non-diabetic) using Chi square test

Association between ophthalmic artery parameter, central retinal artery and central retinal vein (PSV, EDV, PI, RI) and HbA1c variables was assessed by calculating spearman rank correlation coefficient and the data was represented in a scatter diagram.

P value < 0.05 was considered statistically significant. IBM SPSS version 22 was used for statistical analysis.

7. OBSERVATION AND RESULTS:

A total of 213 subjects were included in the analysis.

Table 7.1: Descriptive analysis of group in the study population (N=213)

Study group	Frequency	Percentages
Diabetic retinopathy	55	25.80%
Diabetic without retinopathy	68	31.90%
Non diabetic	90	42.30%

A total of 55 diabetic retinopathy,68 diabetics without retinopathy and 90 non diabetic were included in the final analysis (Table: 7.1 & figure 7.1)

Figure 7. 1: Pie chart of study group in the study population (N=213)

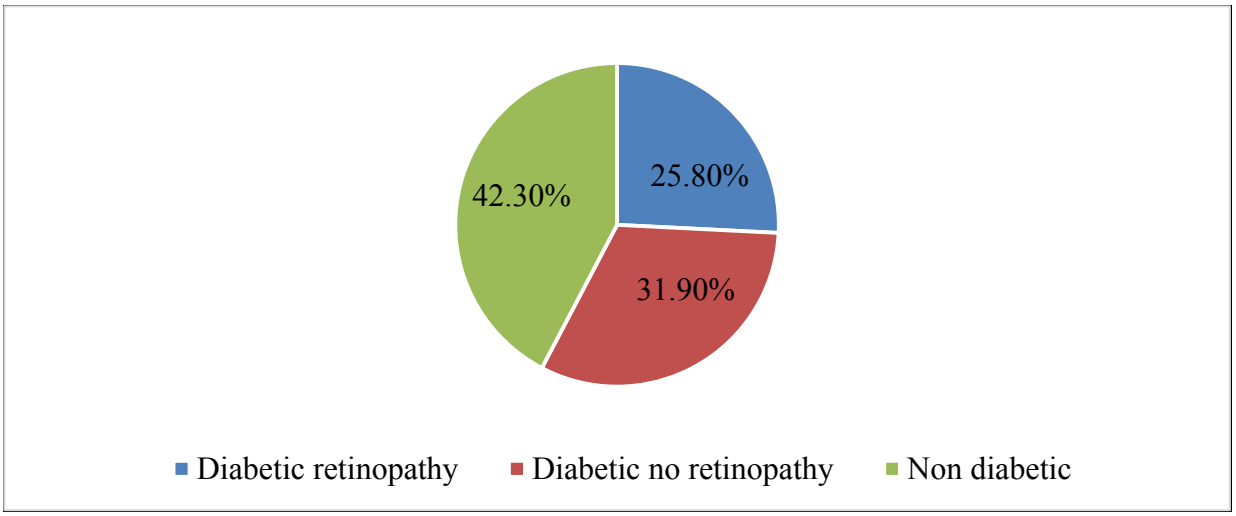


Table 7. 2: Comparison of median values in age across the study group (N=213)

Parameter	Study group Median (IQR)			Kruskal Wallis test (P value)
	Diabetic with retinopathy	Diabetic Without retinopathy	Non diabetic	
Age (in years)	53 (48 to 60)	52 (40.50 to 60)	50 (45 to 56)	0.139

Among the people with diabetic retinopathy median age was 53 years (IQR 48 to 60), the median age of 52 years (IQR 40.50 to 60) among diabetic without retinopathy group, and 50 years (IQR 45 to 56) among non-diabetic. The difference in the median age across study group was statistically not significant (P value 0.139).

Figure 7.2: Bar chart of comparison of median age across the study group (N=213)

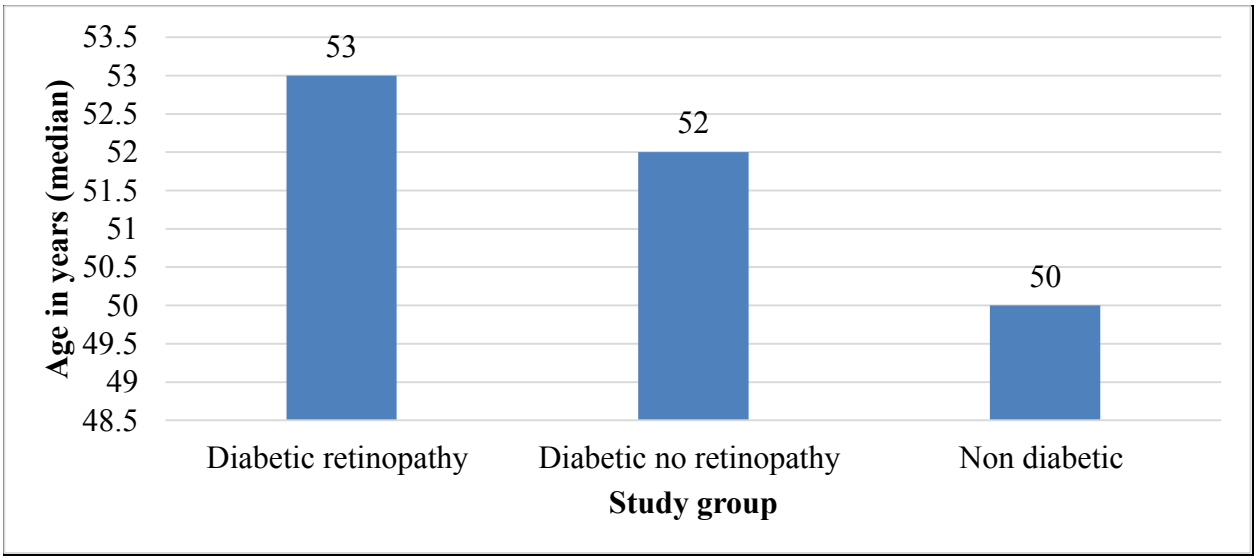


Table7. 3: Comparison of study group with gender (N=213)

Gender	Study group			Chi square	P-value
	Diabetic retinopathy (N=55)	Diabetic without retinopathy (N=68)	Non diabetic (N=90)		
Male	34 (61.8%)	39 (57.4%)	45 (50%)	2.084	0.353
Female	21 (38.2%)	29 (42.6%)	45 (50%)		

Among the people with diabetic retinopathy, 34(61.8%) participants were males and 21(38.2%) participants were females. Among the diabetic non-retinopathy group, 39 (57.4%) participants were males and 29 (42.6%) participants were females. Among non-diabetic population, 45 (50%) participants were males and 45 (50%) participants were females, the difference in the proportion of gender across study group was statistically not significant (p value 0.353).

Figure 7.3: Cluster bar chart of comparison of study group with gender (N=213)

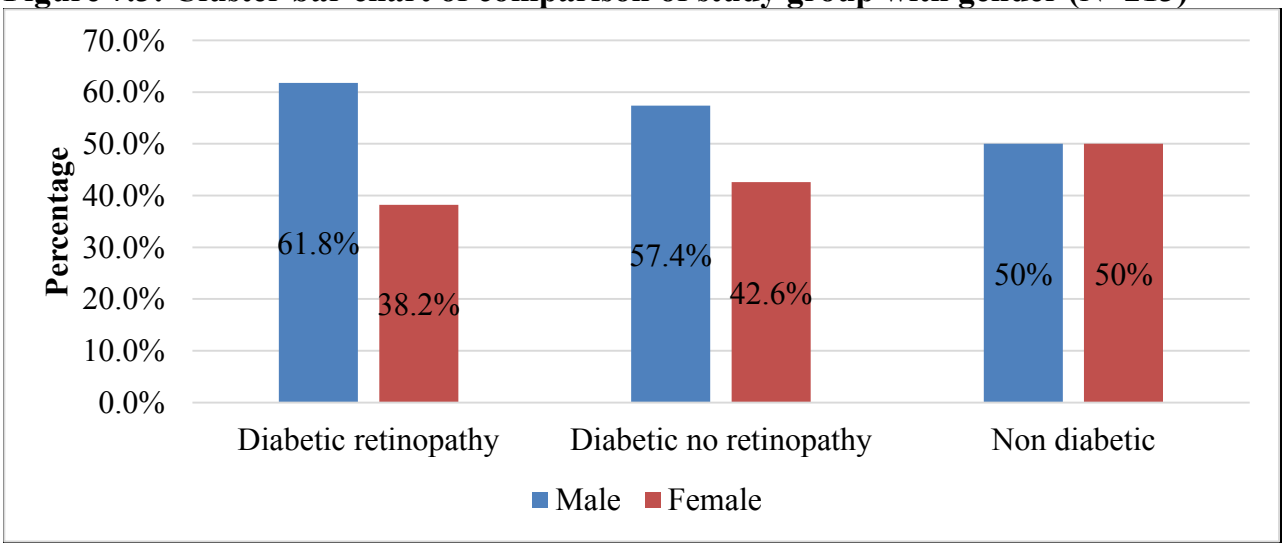


Table7.4: Comparison of anthropometric parameters across the study group (N=213)

Anthropometric parameter	**Diabetic retinopathy** Median (IQR)	**Diabetic** without **retinopathy** Median (IQR)	**Non diabetic** Median (IQR)	**Kruskal Wallis test (P value)**
Height (in cm)	164 (161 to 168)	163(160 to 168)	163(158.75 to 165)	0.036
Weight (in kg)	62(58 to 66)	61.50(56 to 68)	60(54.75 to 64)	0.184

Among the people with study group, diabetic retinopathy median height was 164 cm (IQR 161 to 168), it was 163 cm (IQR 160 to 168) among diabetic without retinopathy group, and 163 cm (IQR 158.75 to 165) among non-diabetic group. The difference in the median height across study group was statistically significant (P value 0.036). Among diabetic retinopathy group median weight was 62 kg (IQR 58 to 66), it was 61.50 kg (IQR 56 to 68) among diabetic without retinopathy, and 60 kg (IQR 54.75 to 64) among non-diabetic population. The difference in the median weight across study group was statistically not significant (P value 0.184).

Figure 7.4: Bar chart of comparison of median height across the study group (N=213)

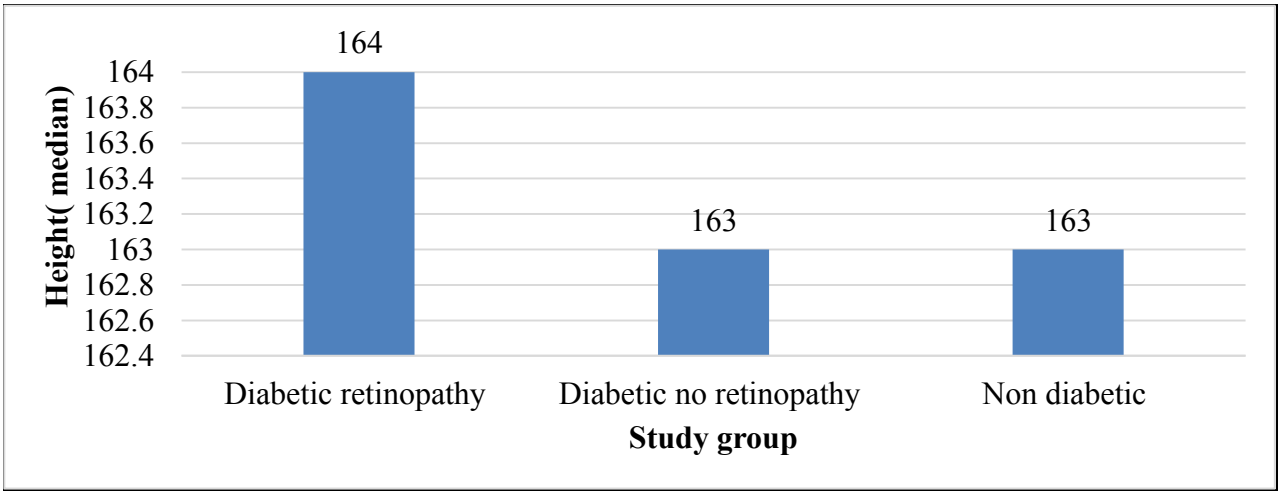


Figure 7. 5: Bar chart of comparison of median weight across the study group (N=213)

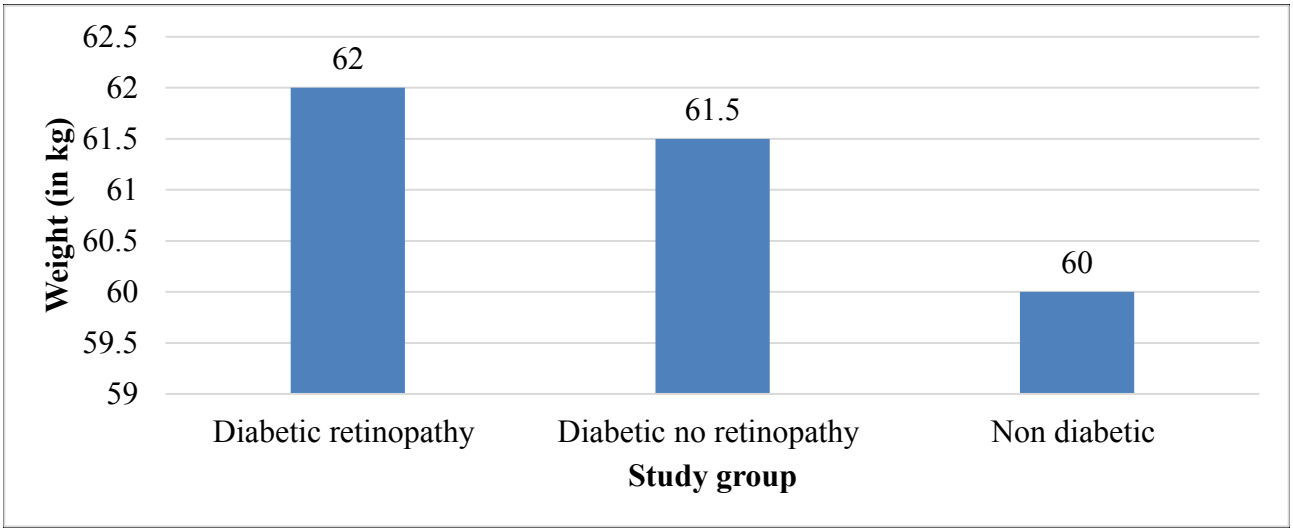


Table 7.5: Comparison of median values in blood pressure across the study group (N=213)

Blood pressure	**Diabetic retinopathy** Median (IQR)	**Diabetic without retinopathy** Median (IQR)	**Non diabetic** Median (IQR)	**Kruskal Wallis test (P value)**
Systolic blood pressure	118 (110 to 120)	118(110 to 120)	110(110 to 120)	0.009
Diastolic blood pressure	80(70 to 80)	80(70 to 80)	72(70 to 72)	0.102

Among, diabetic retinopathy group, median Systolic blood pressure was 118 mm Hg (IQR 110 to 120), it was 118 mm Hg (IQR 110 to 120) among diabetic without retinopathy, and 110mm hg (IQR 110 to 120) among non-diabetic group. The difference in the median systolic blood pressure across study group was statistically significant (P value 0.009).

Among the people with diabetic retinopathy median diastolic blood pressure was 80mmhg (IQR 70 to 80) , It was 80 mm Hg (IQR 70 to 80) among diabetic without retinopathy, and 72 mm Hg (IQR 70 to 72) among non-diabetic. The difference in the median diastolic blood pressure across study group was statistically not significant (P value 0.102).

Figure 7. 6: Bar chart of comparison of median systolic blood pressure across the study group (N=213)

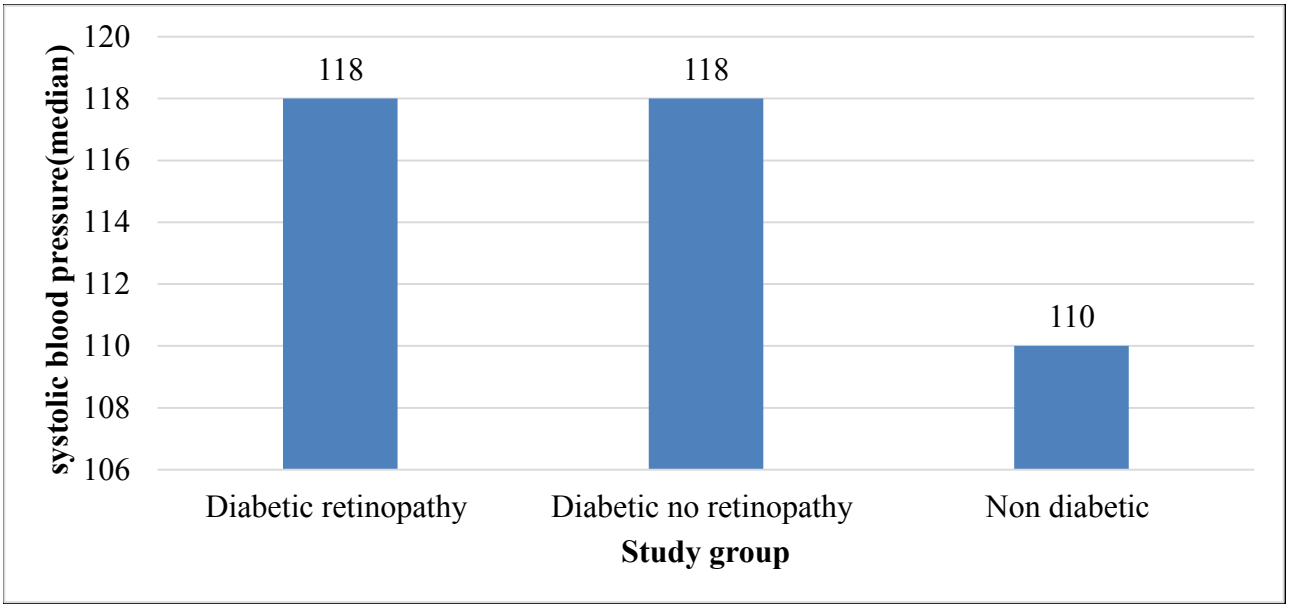


Figure 7.7: Bar chart of comparison of median diastolic blood pressure across the study group (N=213)

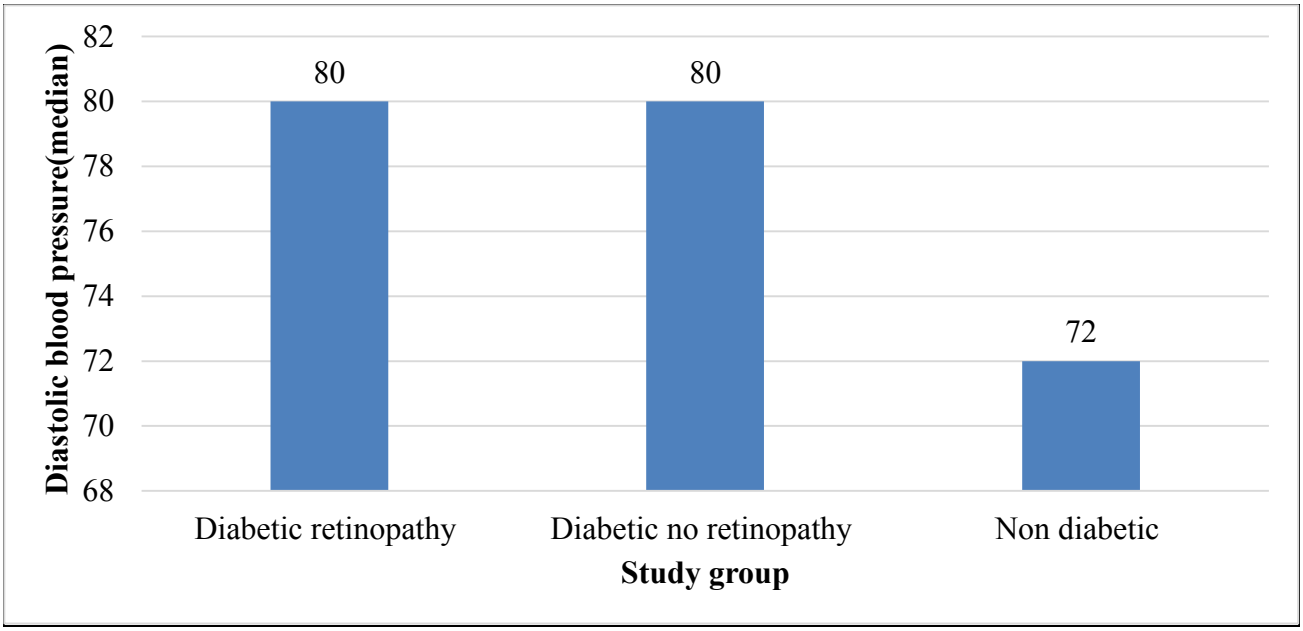


Table 7.6: Comparison of median values in blood test across the study group (N=213)

Blood test	**Diabetic retinopathy** Median (IQR)	**Diabetic without retinopathy** Median (IQR)	**Non diabetic** Median (IQR)	**Kruskal Wallis test (P value)**
Fasting blood sugar	260(199 to 311)	232 (193.50 to 282.25)	91(84 to 99.50	<0.001
Post prandial sugar	308(256 to 380)	282.50(246 to 342)	123(113 to 128)	<0.001

Among the people with diabetic retinopathy median fasting blood sugar was 260 gm/dl (IQR 119 to 311), it was 232 gm/dl (IQR 193.50 to 282.25) among diabetic without retinopathy group, and 91gm/dl (IQR 84 to 99.50) among non-diabetic. The difference in the median fasting blood sugar across study group was statistically significant (P value <0.001). Among the people with diabetic retinopathy median post prandial sugar was 308 gm/dl (IQR 256 to 380), it was 282.50 gm/dl (IQR 256 to 380) among diabetic without retinopathy, and 123 (IQR 113 to 128) among non-diabetic. The difference in the median post prandial sugar across study group was statistically significant (P value <0.001).

Figure 7.8: Bar chart of comparison of median fasting blood sugar across the study group(N=213)

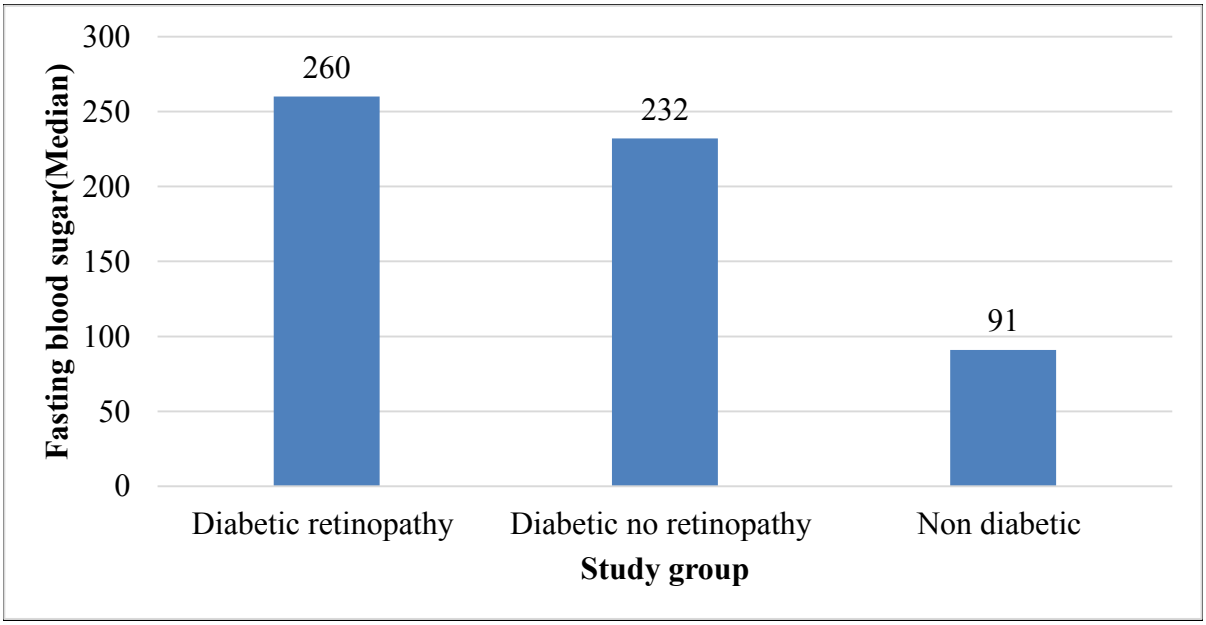


Figure7. 9: Bar chart of comparison of median post prandial blood sugar across the study group (N=213)

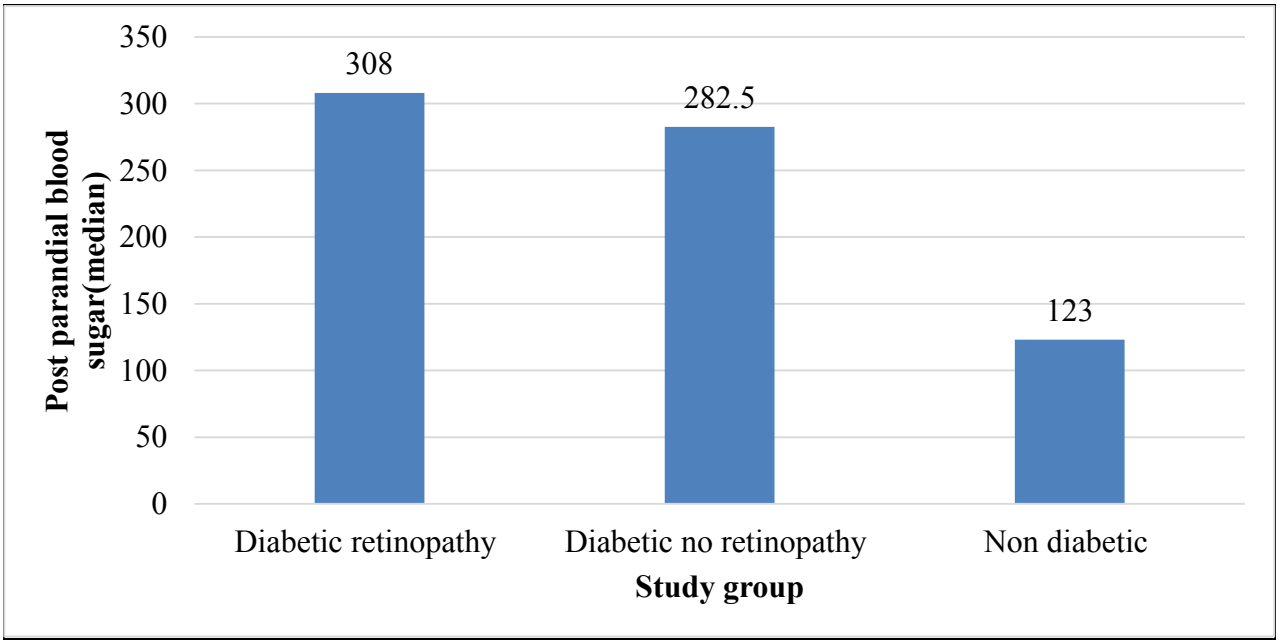


Table7.7: Comparison of median values in HbA1c across the study group (N=213)

Parameter	Diabetic retinopathy Median (IQR)	Diabetic without retinopathy Median (IQR)	Non diabetic Median (IQR)	Kruskal Wallis test (P value)
HbA1c	8.20(7.80 to 9)	7.40 (7.10 to 7.60)	4.90(4.50 to 5.10)	<0.001

Among the people with study group, diabetic retinopathy median HbA1c was 8.20(IQR 7.80 to 9), it was 7.40 (IQR 7.10 to 7.60) among diabetic without retinopathy group and 4.90 (IQR 4.50 to 5.10) among non-diabetic. The difference in the median HbA1c across study group was statistically significant (P value <0.001).

Figure 7.10: Bar chart of comparison of median HbA1c across the study group (N=213)

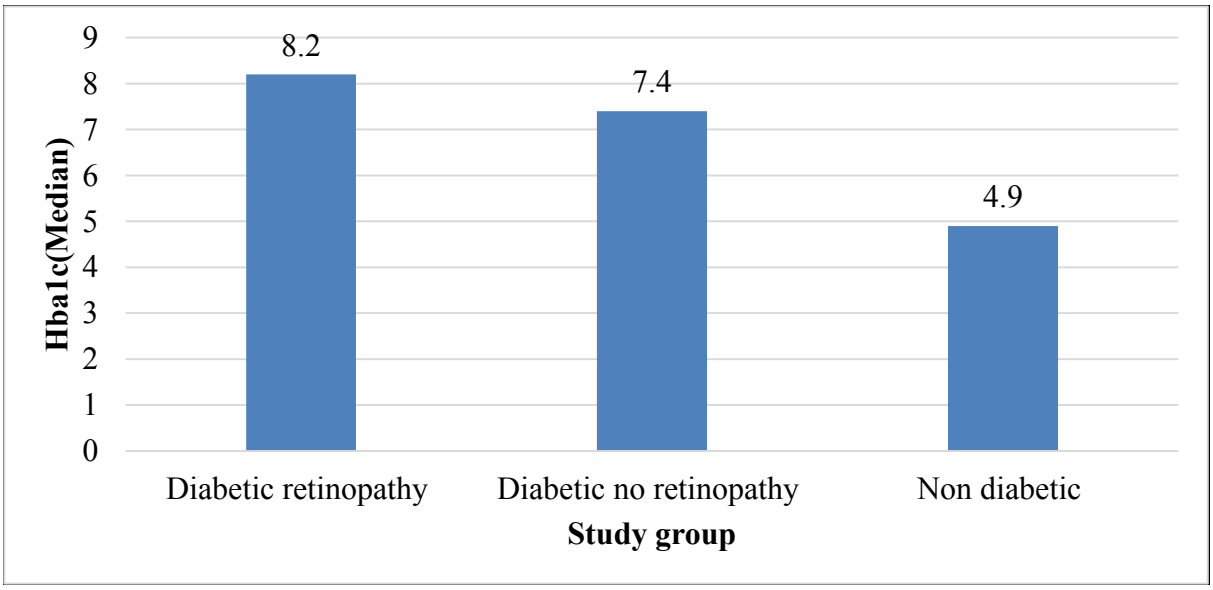


Table 7.8: Comparison of ophthalmic artery parameters across the study group (N=213)

Ophthalmic artery	**Diabetic retinopathy** Median (IQR)	**Diabetic without retinopathy** Median (IQR)	**Non diabetic** Median (IQR)	**Kruskal Wallis test (P value)**
PSV	32.90 (27.80 to 40.50)	31.25 (25.92 to 34.57)	30.90(29.67 to32.52)	0.347
EDV	5.20(4.10 to 7.40)	6.85(5.12 to8.47)	7.50(7.05 to 8.90)	<0.001
PI	2.04(1.73 to 2.30)	1.65 (1.45 to 1.87)	1.52 (1.36 to 1.55)	<0.001
RI	0.83 (0.79 to 0.87)	0.77(0.73 to 0.80)	0.75 (0.72 to 0.76)	<0.001

Among, diabetic retinopathy group, median PSV of **ophthalmic artery** was 32.90 (IQR 27.80 to 40.50) , it was 31.25 (IQR 25.92 to 34.57) among diabetic without retinopathy group, and 30.90 (IQR 29.67 to 32.52) among non-diabetic. The difference in the median PSV across study group was statistically not significant (P value 0.347).

Among, diabetic retinopathy group, median EDV of **ophthalmic artery** was 5.20 (IQR 4.10 to 7.40), it was 6.85 (IQR 5.12 to 8.47) among diabetic without retinopathy, and 7.50 (IQR 7.05 to 8.90) among non-diabetic. The difference in the median EDV across study group was statistically significant (P value <0.001).

Among, diabetic retinopathy group, median PI of **ophthalmic artery** was 2.04 (IQR 1.73 to 2.30), it was 1.65 (IQR 1.45 to 1.87) among diabetic non-retinopathy, and 1.52 (IQR 1.36 to 1.55) among non-diabetic. The difference in the median PI across study groups was statistically significant (P value <0.001).

Among, diabetic retinopathy group, median RI of **ophthalmic artery** was 0.83 (IQR 0.79 to 0.87), it was 0.77 (IQR 0.73 to 0.80) among diabetic without retinopathy, and 0.75 (IQR 0.72 to 0.76) among non-diabetic. The difference in the median RI across study groups was statistically significant (P value <0.001)

Figure 7.11: Bar chart of comparison of median OA PSV across the study group (N=213)

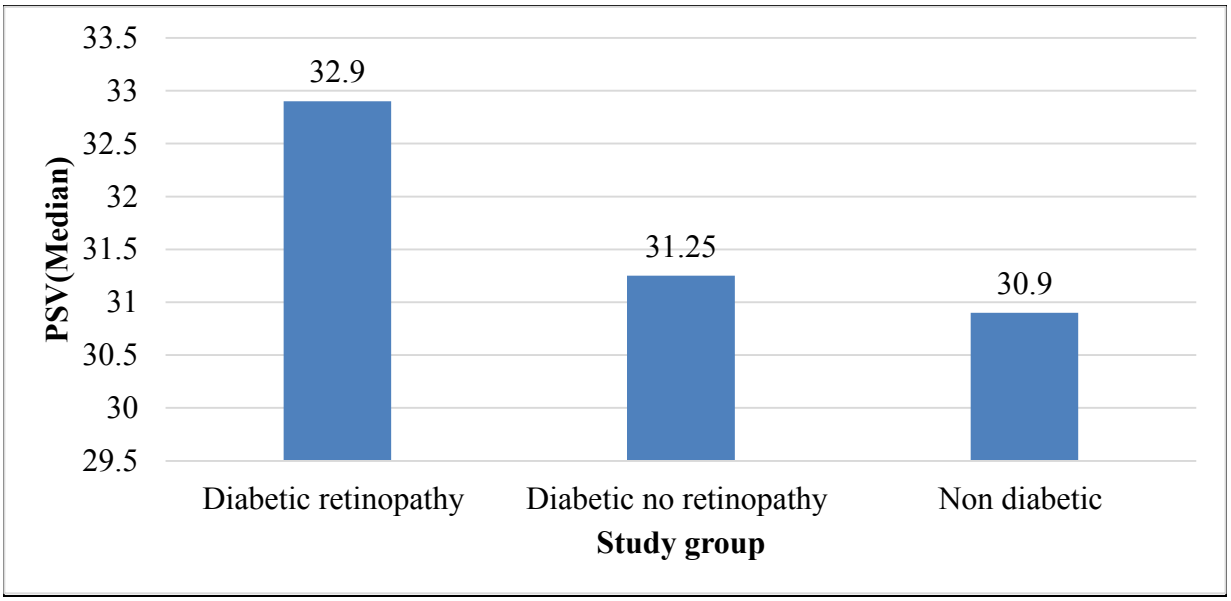


Figure7. 12: Bar chart of comparison of median OA EDV across the study group (N=213)

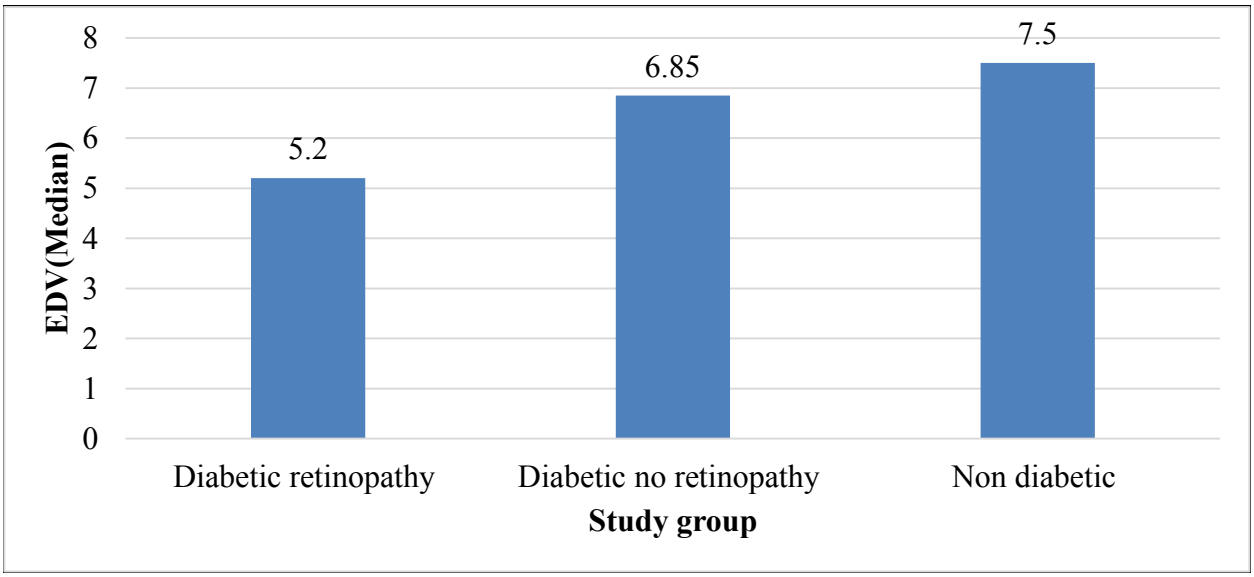


Figure7. 13: Bar chart of comparison of median OA PI across the study group (N=213)

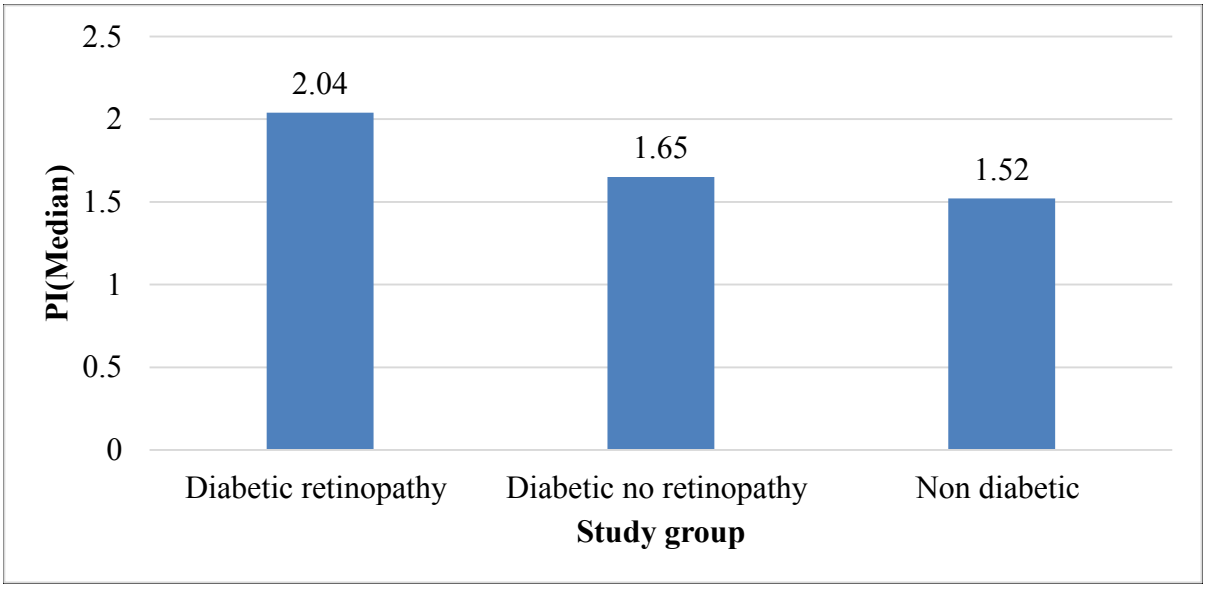


Figure 7.14: Bar chart of comparison of median OA RI across the study group (N=213)

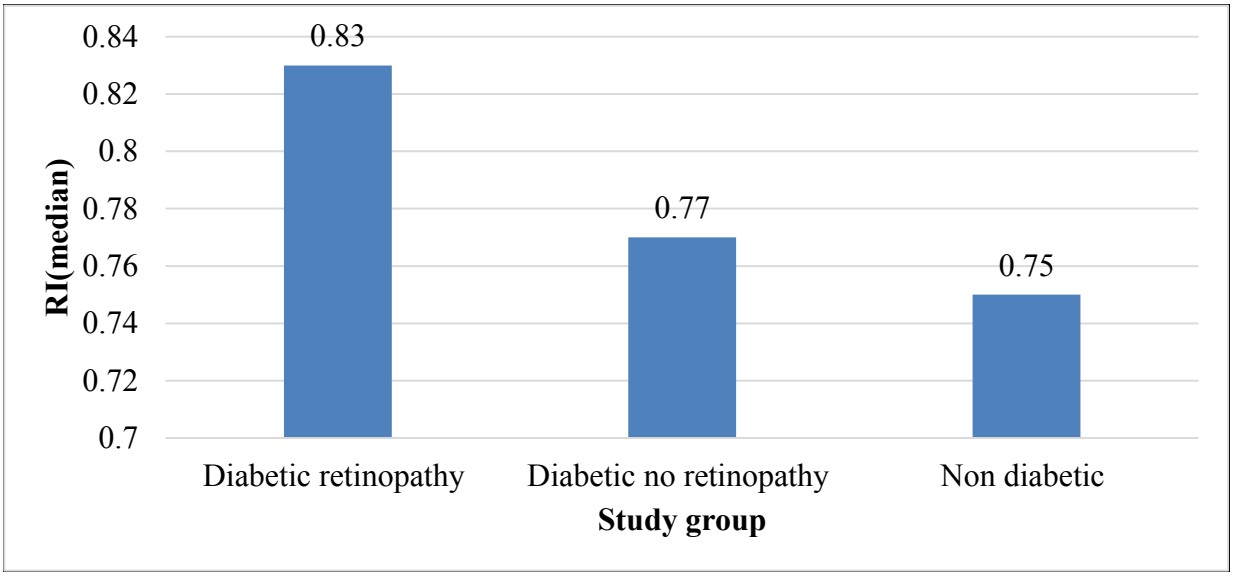


Table7.9: Comparison of central retinal artery parameters across the study group (N=213)

Central retinal artery	**Diabetic retinopathy** Median (IQR)	**Diabetic** without **retinopathy** Median (IQR)	**Non diabetic** Median (IQR)	**Kruskal Wallis test (P value)**
PSV	11.70 (7.97 to 15.45)	10.15 (8.57 to 14.05)	11(10.10 to 11.85)	0.343
EDV	2.20(1.80 to 3.23)	2.60 (2 to 3.63)	3.50 (2.80 to 4.02)	<0.001
PI	1.48(1.33 to 1.74)	1.46 (1.30 to 1.74)	1.35 (1.22 to 1.55)	<0.001
RI	0.77 (0.73 to 0.84)	0.76(0.71 to 0.82)	0.67 (0.63 to 0.73)	<0.001

Among, diabetic retinopathy group, median PSV of **Central Retinal artery** was 11.70 (IQR 7.97 to 15.45), it was 10.15 (IQR 8.57 to 14.05) among diabetic without retinopathy group and 11 (IQR 10.10 to 11.85) among non-diabetic. The difference in the median PSV of **Central Retinal artery** across study groups was statistically not significant (P value 0.343).

Among, diabetic retinopathy group, median EDV of **Central Retinal artery** was 2.20 (IQR 1.80 to 3.23), it was 10.15 (IQR 8.57 to 14.05) among diabetic without retinopathy, and 11 (IQR 10.10 to 11.85) among non-diabetic population. The difference in the median EDV of **Central Retinal artery** across study groups was statistically significant (P value <0.001).

Among, diabetic retinopathy group, median PI of **Central Retinal artery** was 1.48 (IQR 1.33 to 1.74), it was 1.46 (IQR 1.30 to 1.74) among diabetic without retinopathy, and 1.35 (IQR 1.22 to 1.55) among non-diabetic. The difference in the median PI of **Central Retinal artery** across study groups was statistically significant (P value <0.001).

Among, diabetic retinopathy group, median RI of **Central Retinal artery** was 0.77 (IQR 0.79 to 0.84), it was 0.76 (IQR 0.71 to 0.82) among diabetic without retinopathy, and 0.67 (IQR 0.63 to 0.73) among non-diabetic. The difference in the median RI of **Central Retinal artery** across study groups was statistically significant (P value <0.001)

Figure 7.15: Bar chart of comparison of median CRA PSV across the study group (N=213)

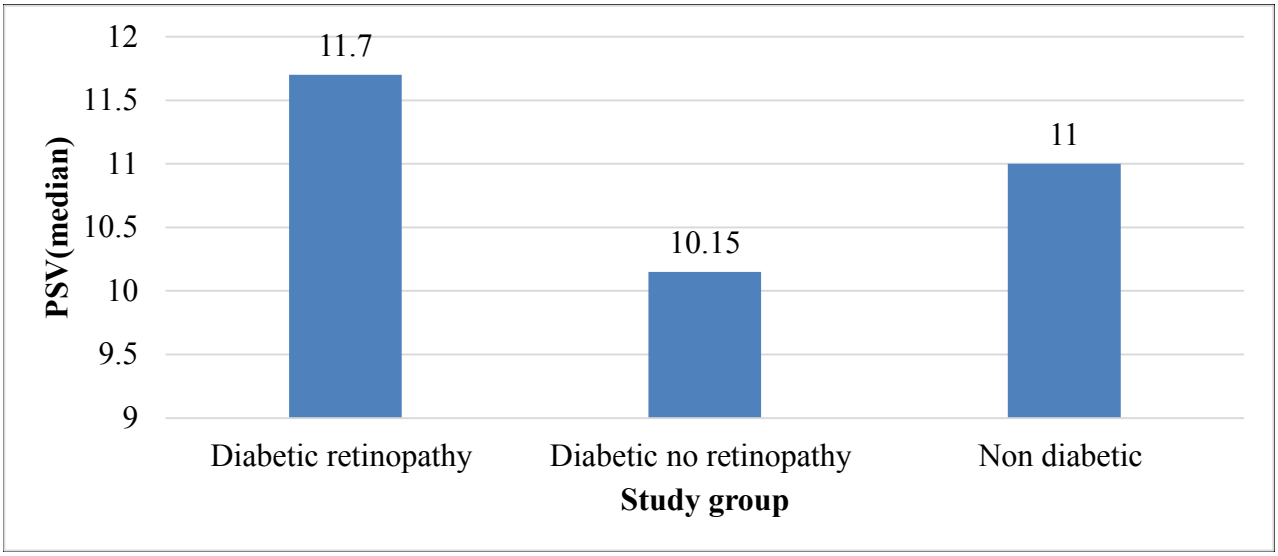


Figure7. 16: Bar chart of comparison of median CRA EDV across the study group (N=213)

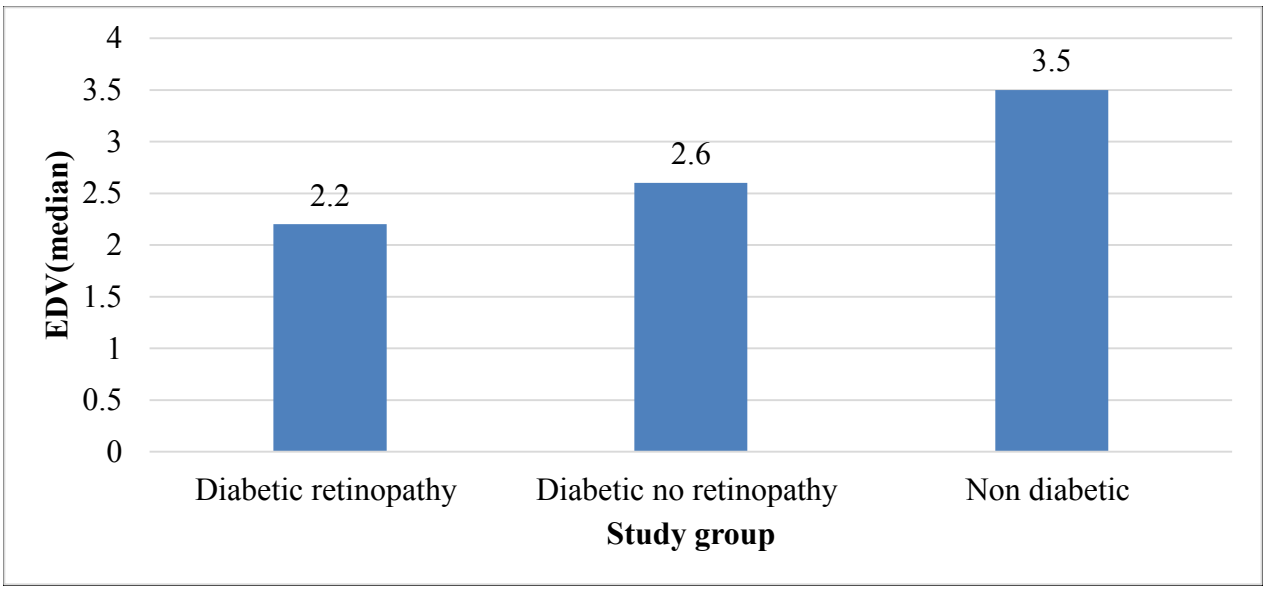


Figure7. 17: Bar chart of comparison of median CRA PI across the study group (N=213)

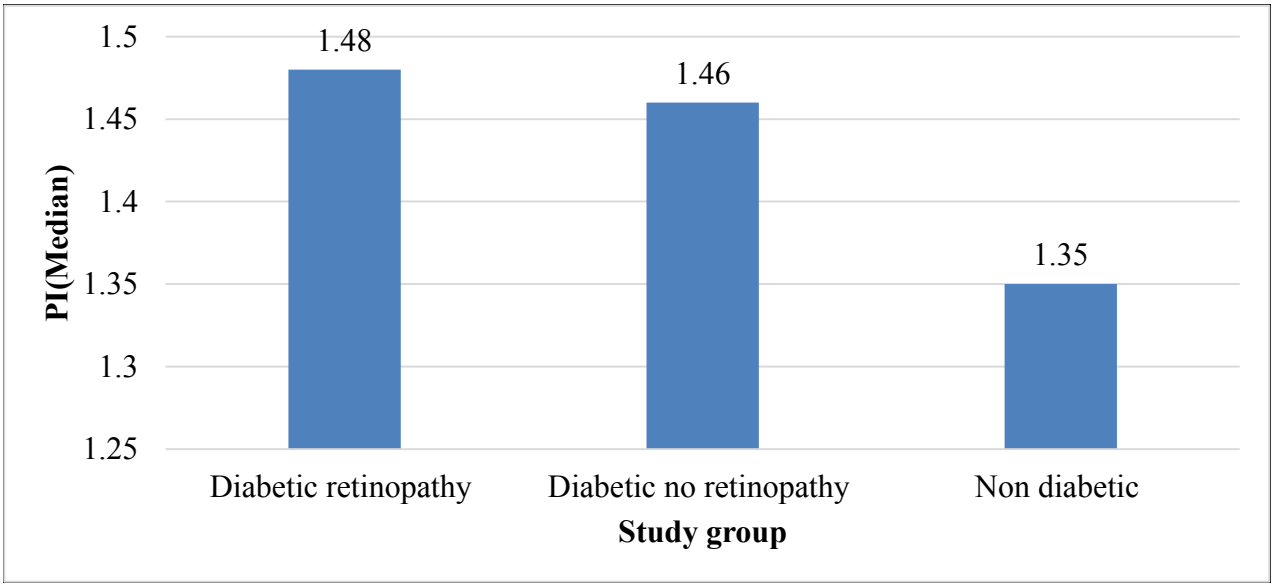


Figure 7.18: Bar chart of comparison of median CRA RI across the study group (N=213)

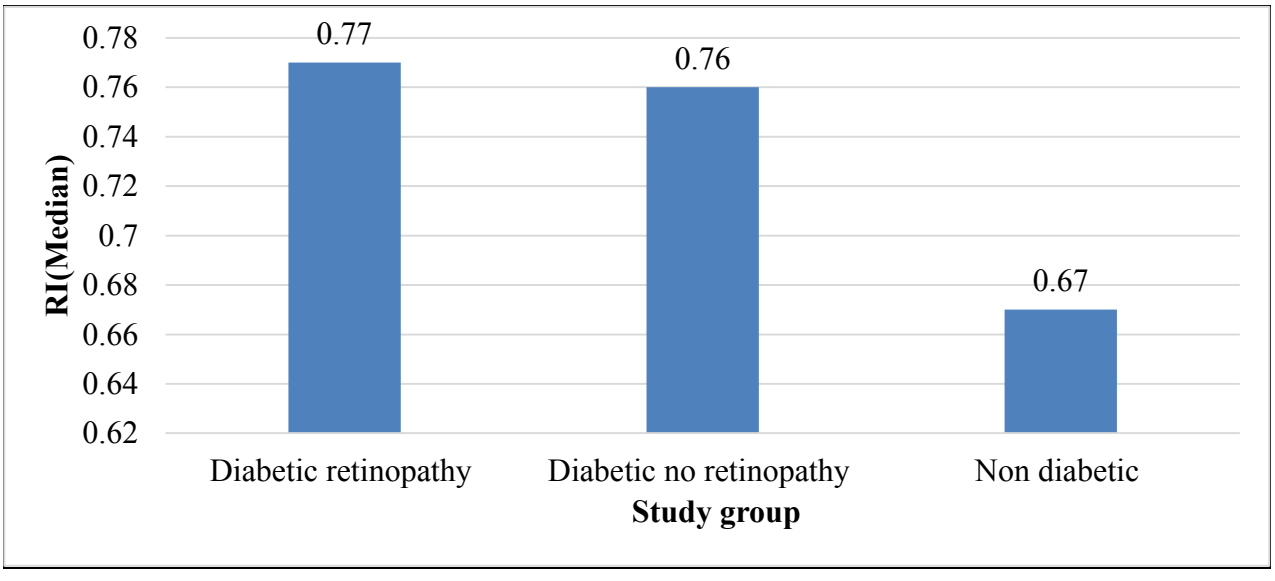


Table7. 10: Comparison of median values in central retinal vein across the study group (N=213)

Central retinal vein	**Diabetic retinopathy** Median (IQR)	**Diabetic** without **retinopathy** Median (IQR)	**Non diabetic** Median (IQR)	**Kruskal Wallis test (P value)**
PSV	8 (6 to 10.10)	7.60 (5.60 to 9.22)	5.50(5 to 8.52)	<0.001
EDV	3.90 (2.90 to 5.20)	3.90 (3 to 4.77)	3.55 (3.17 to 4.32)	0.727
PI	0.74 (0.49 to 0.98)	0.60 (0.43 to 0.88)	0.47 (0.38 to 0.75)	0.001
RI	0.50 (0.39 to 0.60)	0.46(0.35 to 0.58)	0.37 (0.30 to 0.54)	0.002

Among the, diabetic retinopathy group median PSV of **central retinal vein** was 8 (IQR 6 to 10.10), it was 7.60 (IQR 5.60 to 9.22) among diabetic without retinopathy, and 5.50 (IQR 5 to 8.52) among non-diabetic. The difference in the median PSV of **central retinal vein** across study group was statistically significant (P value <0.001).

Among the, diabetic retinopathy group median EDV of **central retinal vein** was 3.90 (IQR 2.90 to 5.20), it was 3.90 (IQR 3 to 4.77) among diabetic without retinopathy, and 3.55 (IQR 3.17 to 4.32) among non-diabetic. The difference in the median EDV of **central retinal vein** across study group was statistically not significant (P value 0.727).

Among the, diabetic retinopathy group median PI of **central retinal vein** was 0.74 (IQR 0.49 to 0.98), it was 0.60 (IQR 0.43 to 0.88) among diabetic without retinopathy, and 0.47 (IQR 0.38 to 0.75) among non-diabetic. The difference in the median PI of **central retinal vein** across study group was statistically significant (P value 0.001)

Among the, diabetic retinopathy group median RI of **central retinal vein** was 0.50 (IQR 0.39 to 0.60), it was 0.46 (IQR 0.35 to 0.58) among diabetic without retinopathy, and 0.37 (IQR 0.30 to 0.5) among non-diabetic. The difference in the median RI of **central retinal vein** across study group was statistically significant (P value 0.002)

Figure 7.19: Bar chart of comparison of median CRV PSV across the study group (N=213)

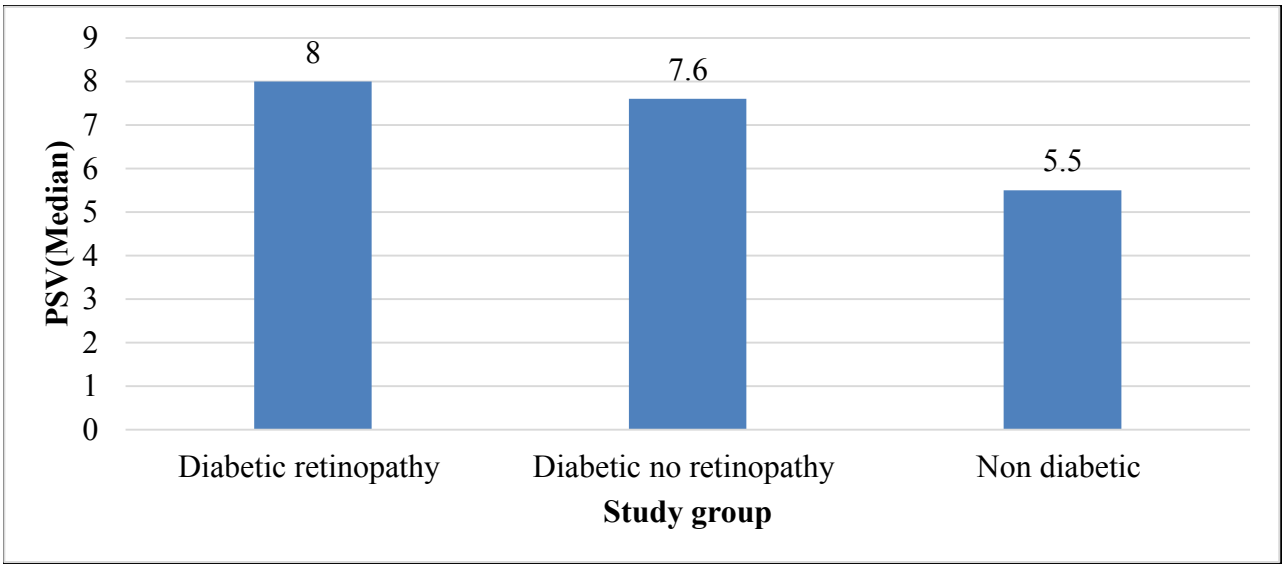


Figure7. 20: Bar chart of comparison of median CRV EDV across the study group (N=213)

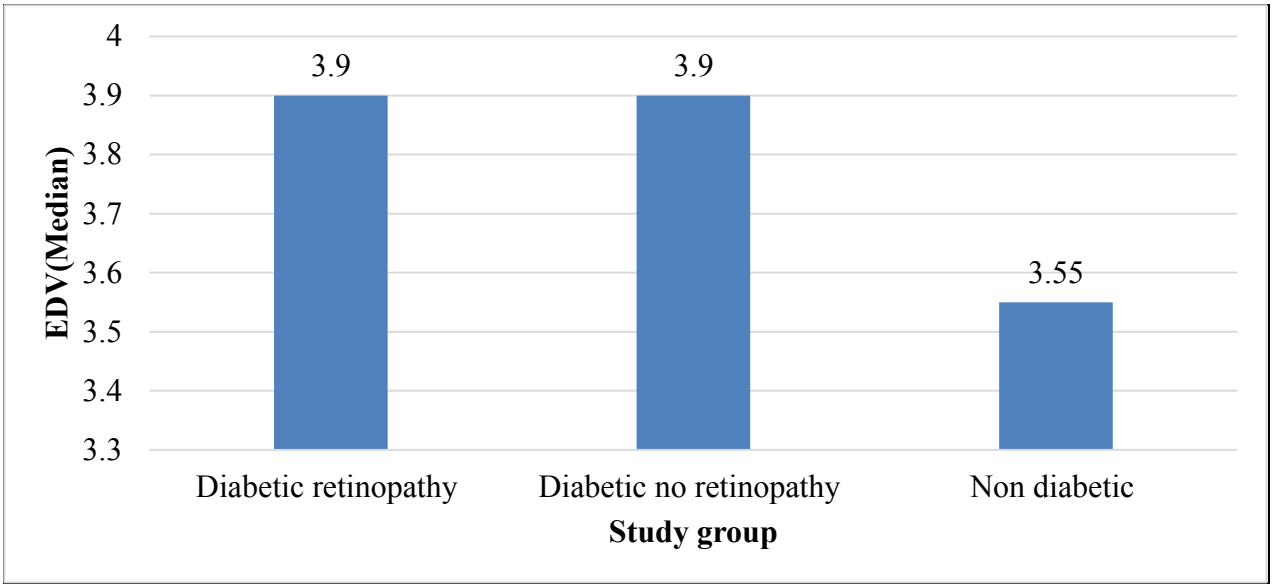


Figure 7.21: Bar chart of comparison of median CRV PI across the study group (N=213)

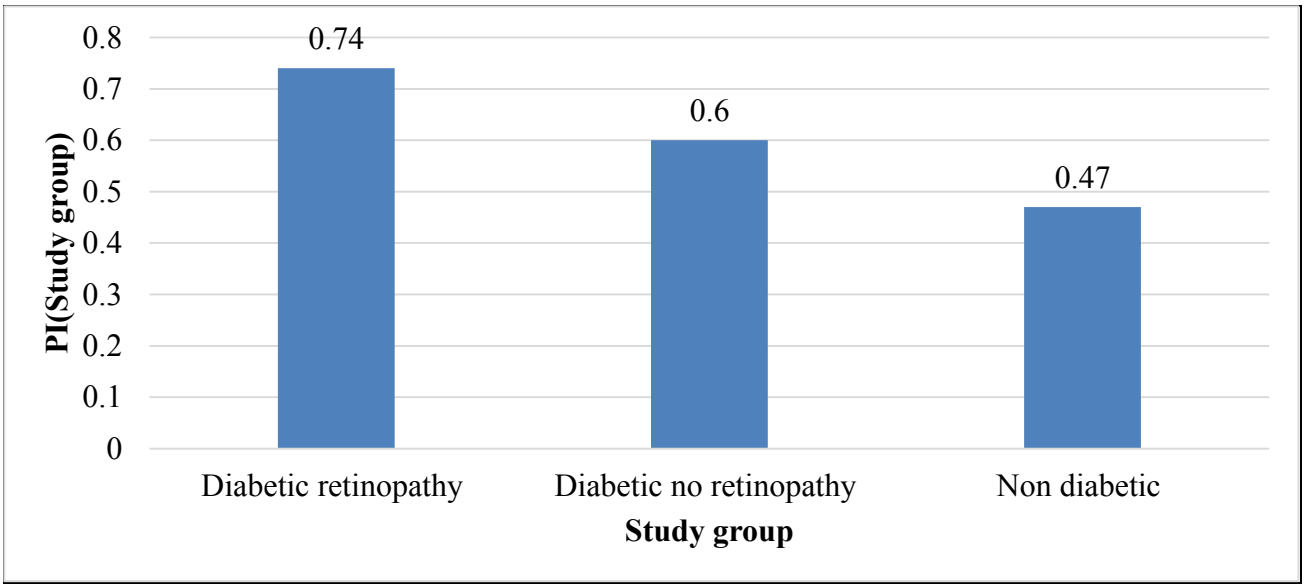


Figure 7.22: Bar chart of comparison of median CRV RI across the study group (N=213)

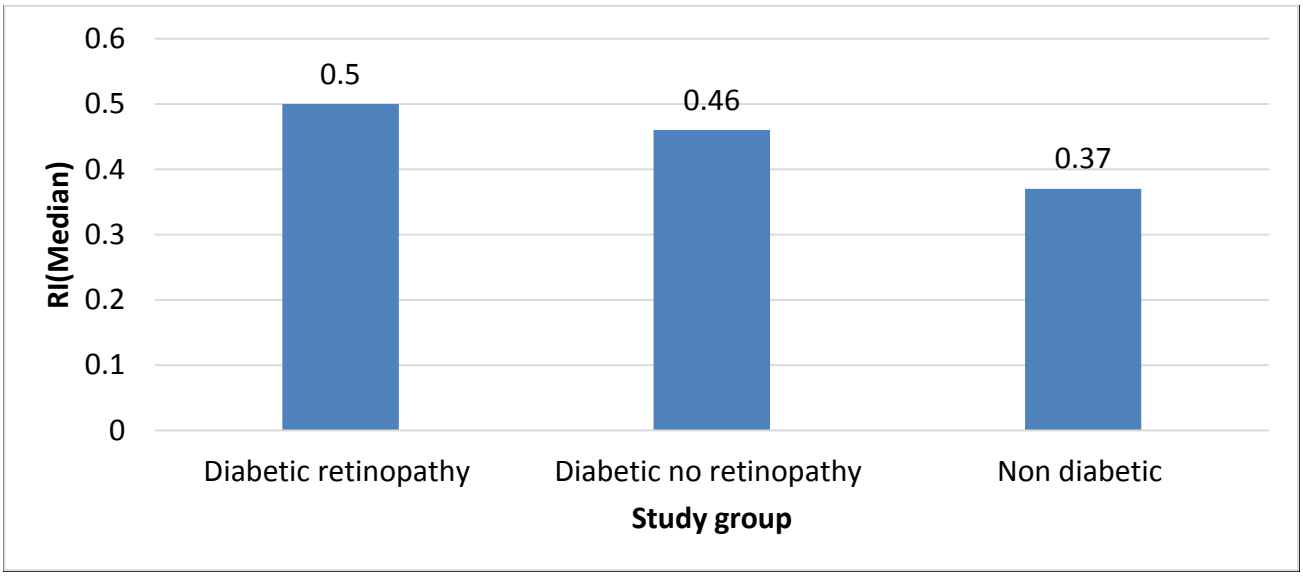


Table7. 11: Comparison of study group with fundos copy (N=213)

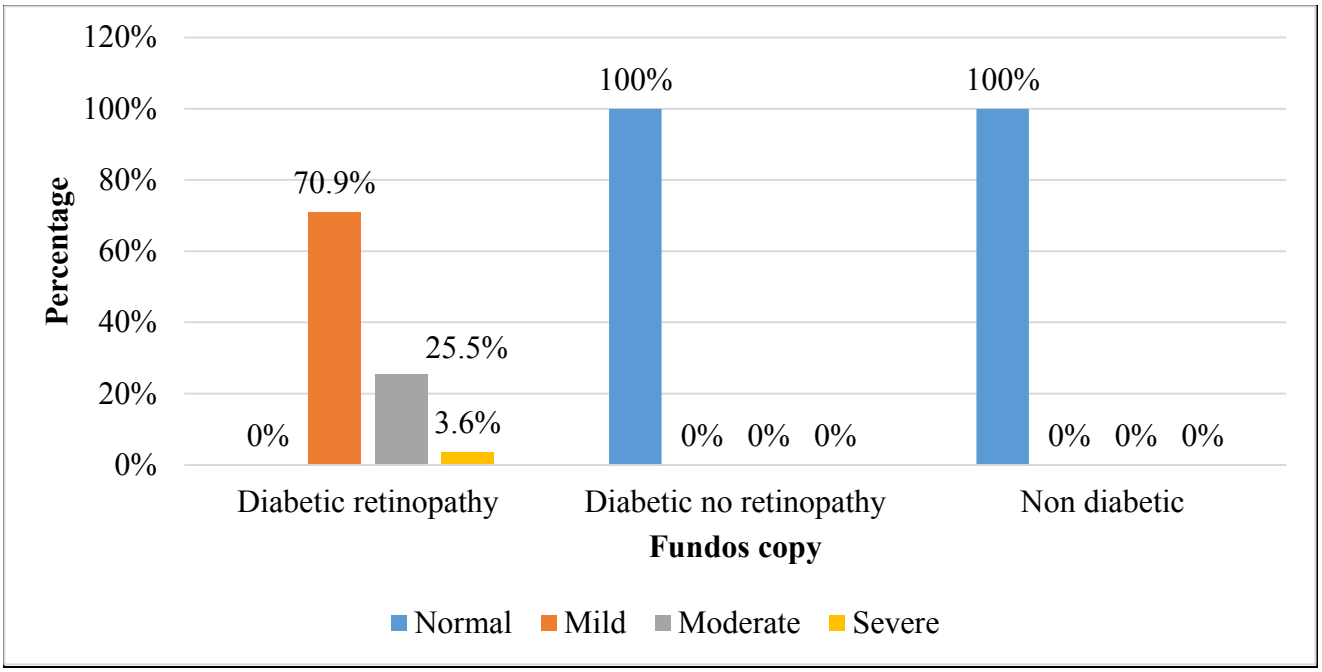
Fundos copy	Study group		
	Diabetic retinopathy (N=55)	**Diabetic** without **retinopathy (N=68)**	**Non diabetic (N=90)**
Normal	0 (0%)	68 (100%)	90 (100%)
Mild	39 (70.9%)	0 (0%)	0 (0%)
Moderate	14(25.5%)	0 (0%)	0 (0%)
Severe	2(3.6%)	0 (0%)	0 (0%)

*No statistical test was applied -due to 0 subjects in the cells

Among the people with diabetic retinopathy, 39 (70.9%) participants had mild 14 (25.5%) participants had moderate and 2(3.6%) participants had severe. Among the people with diabetic

without retinopathy, 68 (100%) participants had normal. Among the people with non -diabetic, 90 (100%) participants had normal.

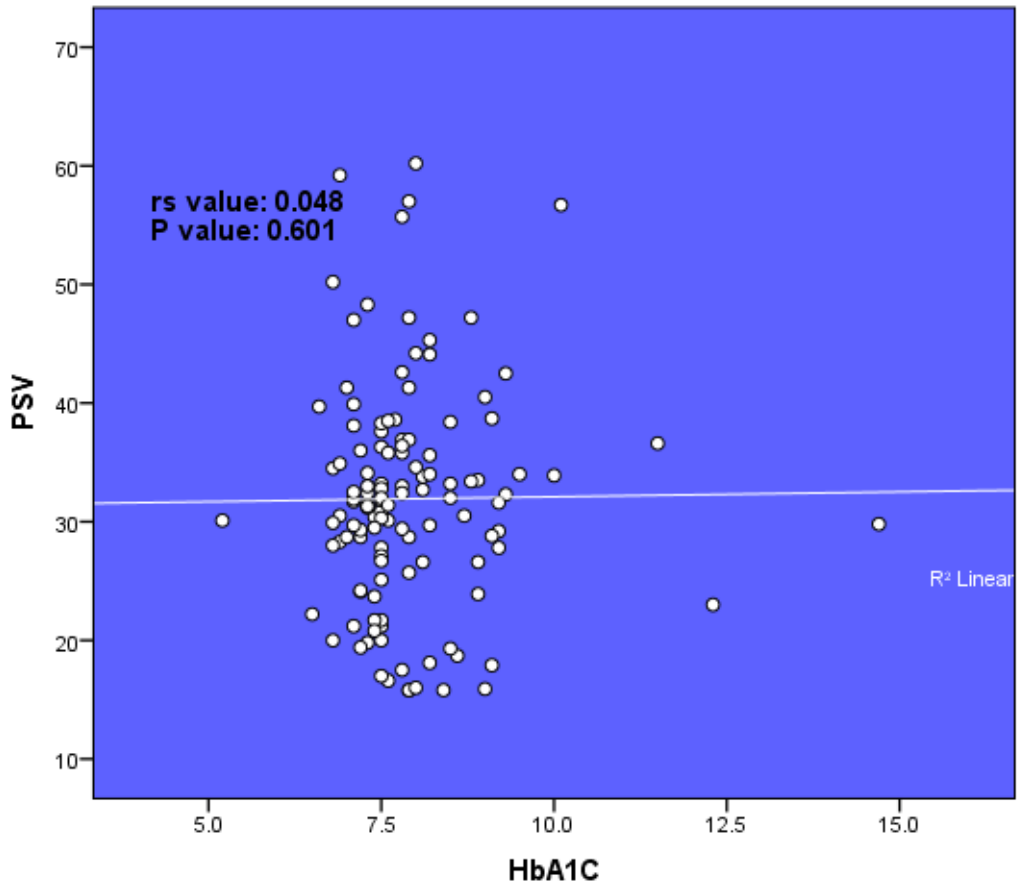
Figure7. 23: Cluster bar chart of comparison of study group with fundoscopy (N=213)



DOPPLER PARAMETER OF OPHTHALMIC ARTERY:

Table 7.12: correlation between HbA1c and ophthalmic artery PSV among diabetic population (N=123)

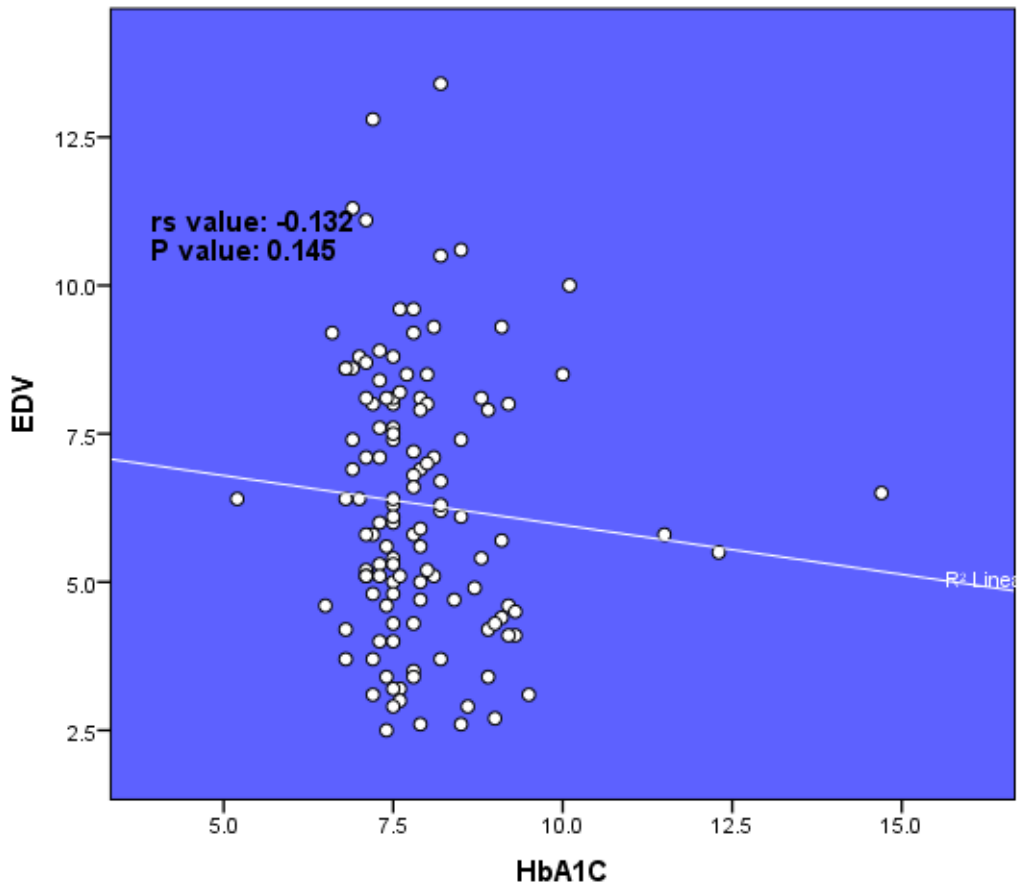
Parameter	Spearman rank Correlation (r_s)	P value
PSV	0.048	0.601



There was a weak positive correlation between HbA1c and PSV (r_s value: 0.048, P value 0.601)

Table7. 13: correlation between HbA1c and ophthalmic artery EDV among diabetic population (N=123)

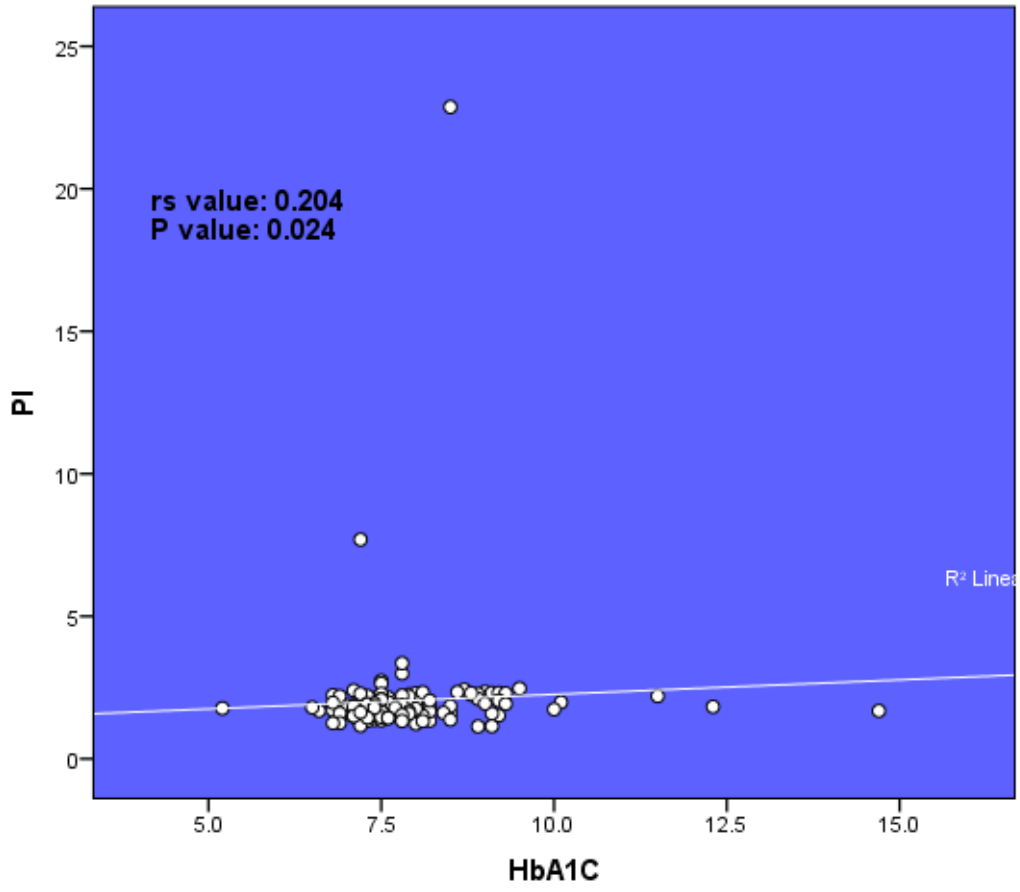
Parameter	Spearman rank Correlation (r_s)	P value
EDV	-0.132	0.145



There was a weak negative correlation between HbA1c and EDV (r_s value: -0.132, P value 0.145)

Table7. 14: correlation between HbA1c and ophthalmic artery PI among diabetic population (N=123)

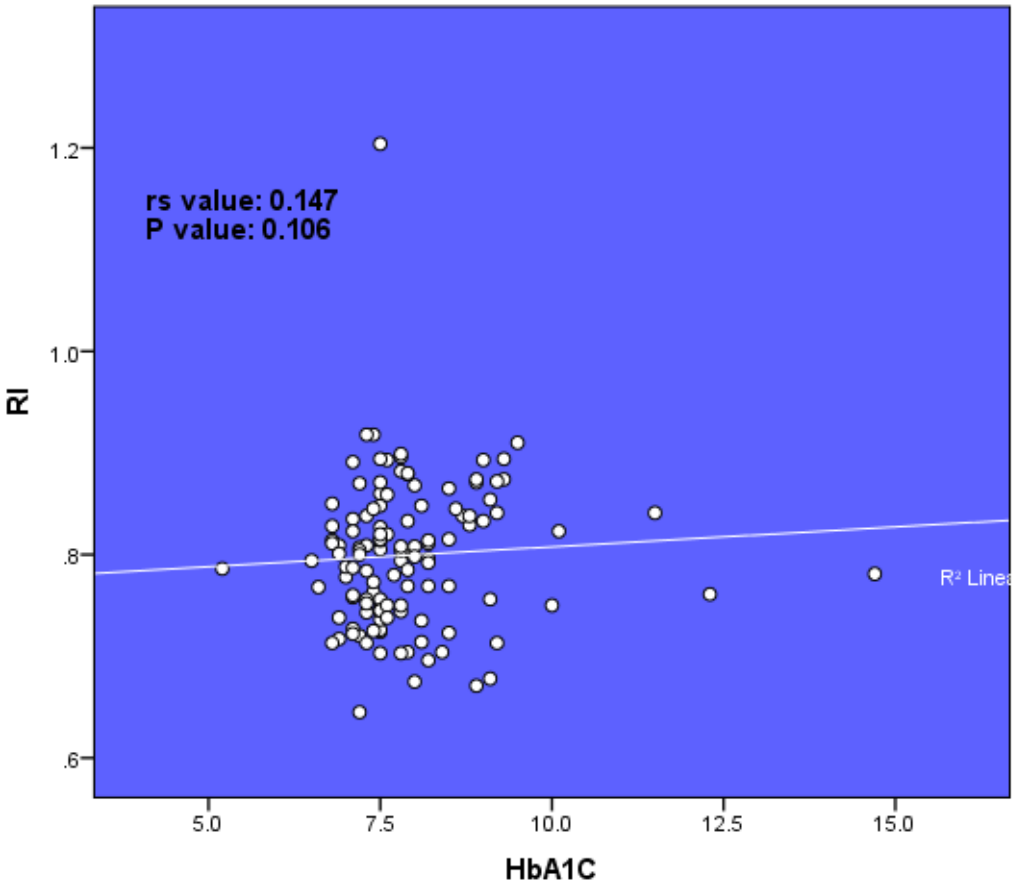
Parameter	Spearman rank Correlation (r_s)	P value
PI	0.204	0.024



There was a weak positive correlation between HbA1c and PI (r_s value: 0.204, P value 0.024)

Table 7.15: correlation between HbA1c and ophthalmic artery RI among diabetic population (N=123)

Parameter	Spearman rank Correlation (r_s)	P value
RI	0.147	0.106

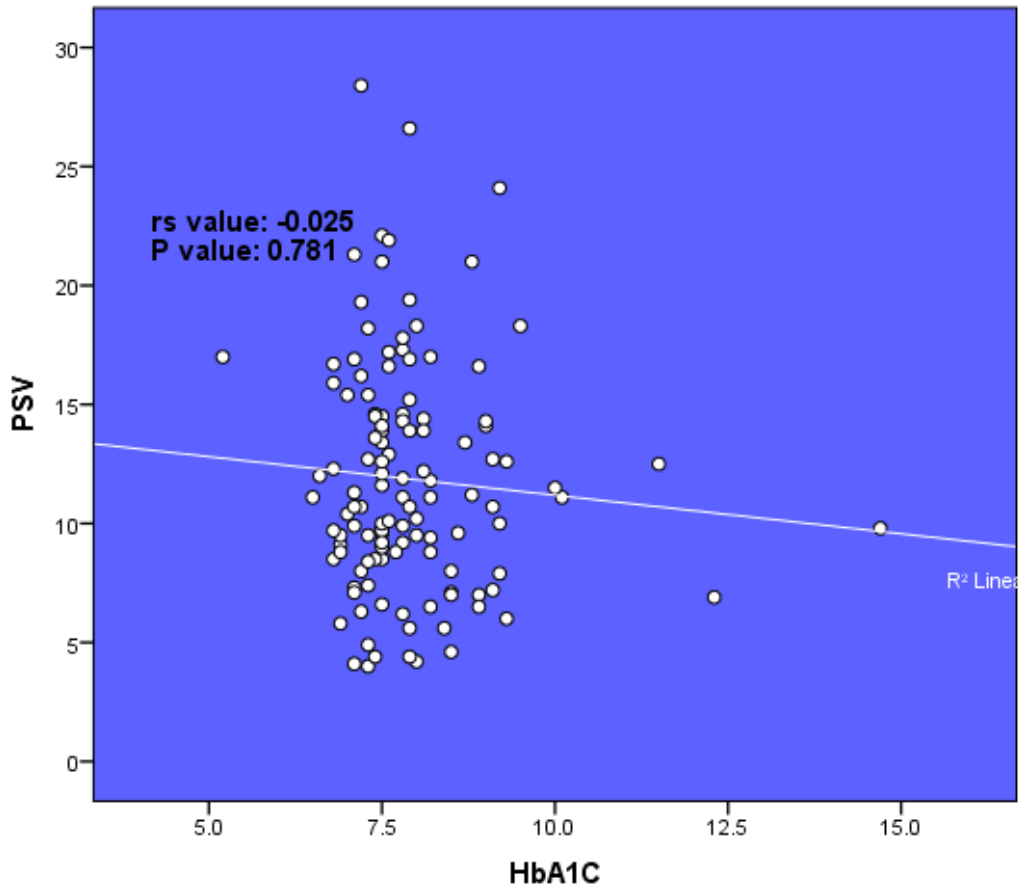


There was a weak positive correlation between HbA1c and RI (r_s value: 0.147, P value 0.106)

DOPPLER PARAMETER OF CENTRAL RETINAL ARTERY:

Table7. 16: correlation between HbA1c and central retinal artery PSV among diabetic population(N=123)

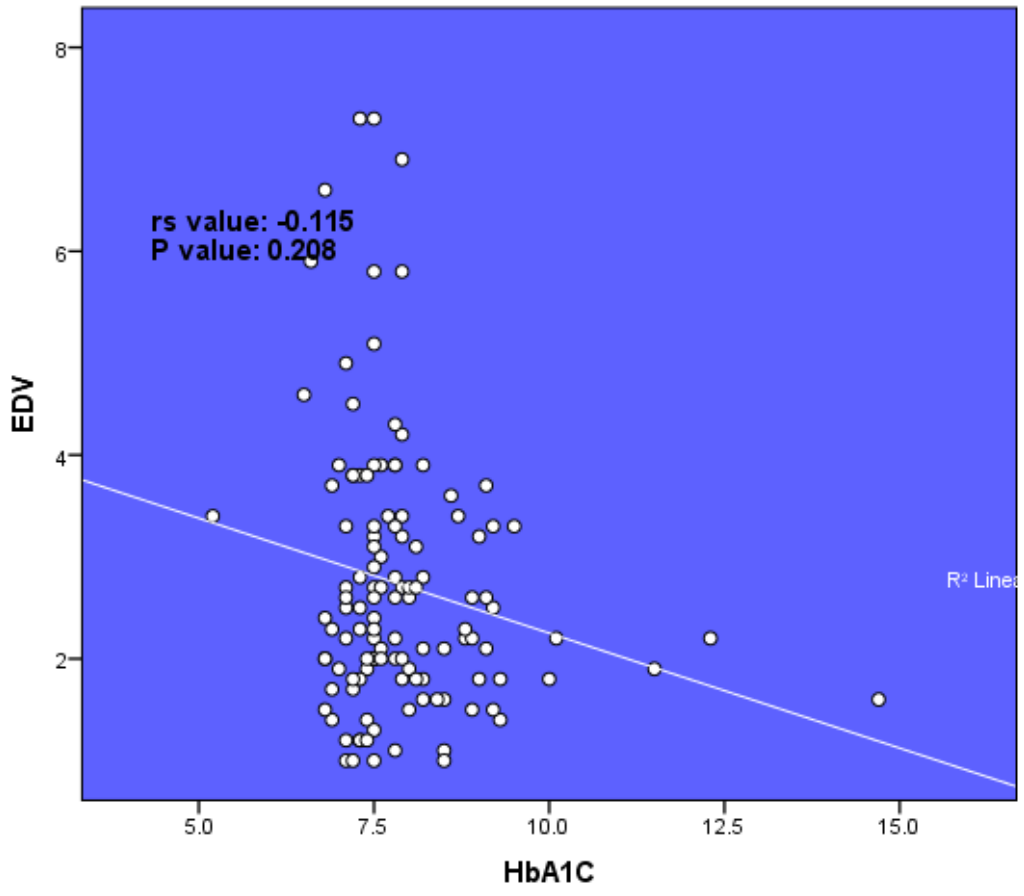
Parameter	Spearman rank Correlation (r_s)	P value
PSV	-0.025	0.781



There was a weak negative correlation between HbA1c and PSV (r_s value: -0.025, P value 0.781)

Table 7.17: correlation between HbA1c and central artery EDV among diabetic population(N=123)

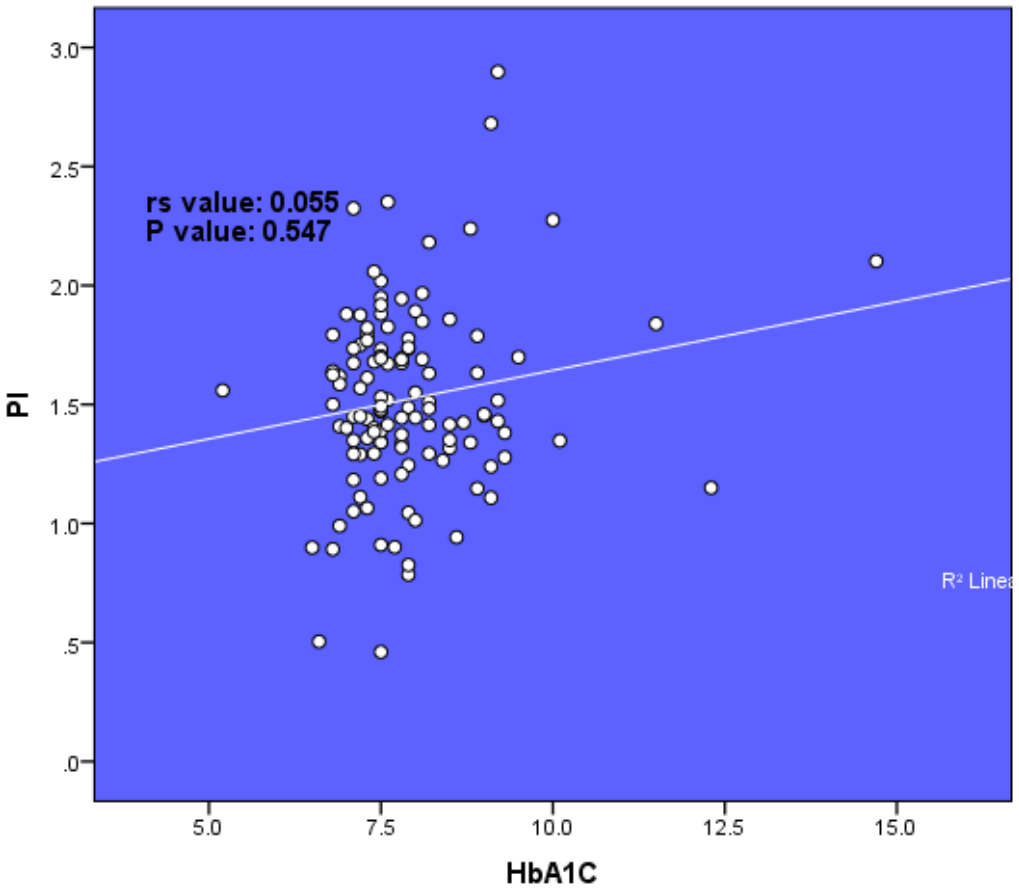
Parameter	Spearman rank Correlation (r_s)	P value
EDV	-0.115	0.208



There was a weak negative correlation between HbA1c and EDV (r_s value: -0.115, P value 0.208)

Table 7.18: correlation between HbA1c and central artery PI among diabetic population (N=123)

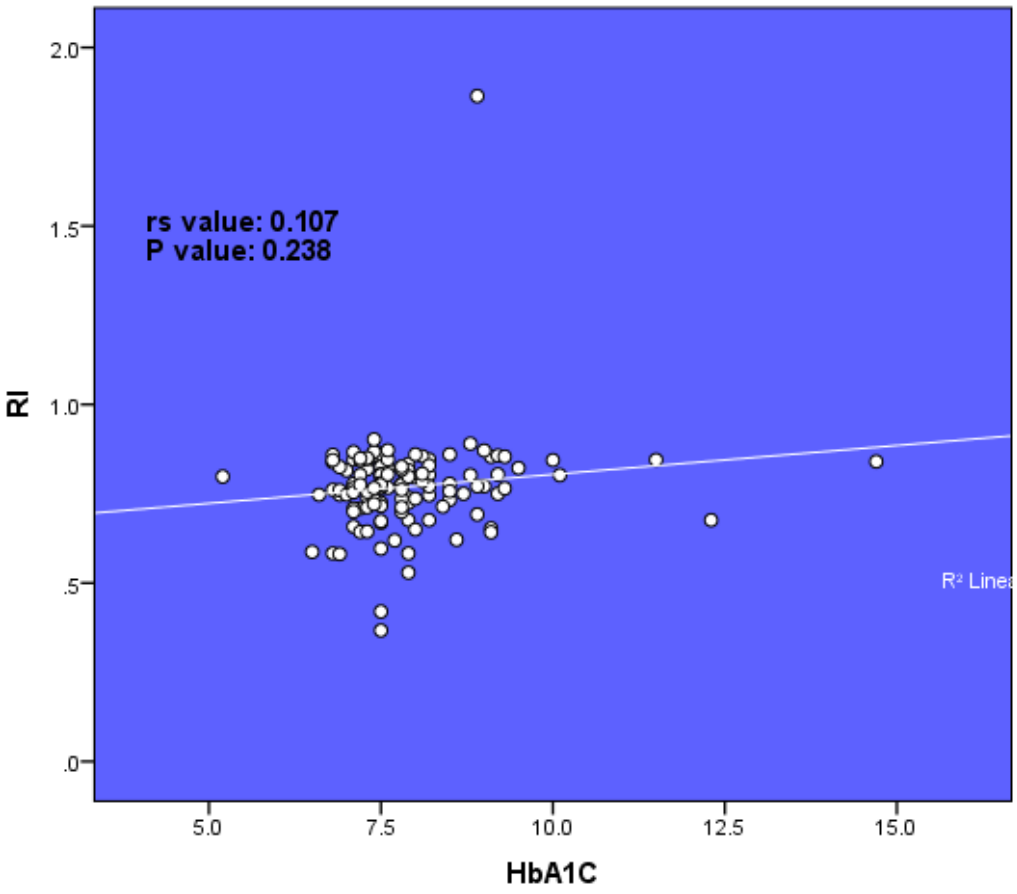
parameter	Spearman rank Correlation (r_s)	P value
PI	0.055	0.547



There was a weak positive correlation between HbA1c and PI (r_s value: 0.055, P value 0.547)

Table7. 19: correlation between HbA1c and Doppler parameters of central artery RI among diabetic population (N=123)

Parameter	Spearman rank Correlation (r_s)	P value
RI	0.107	0.238

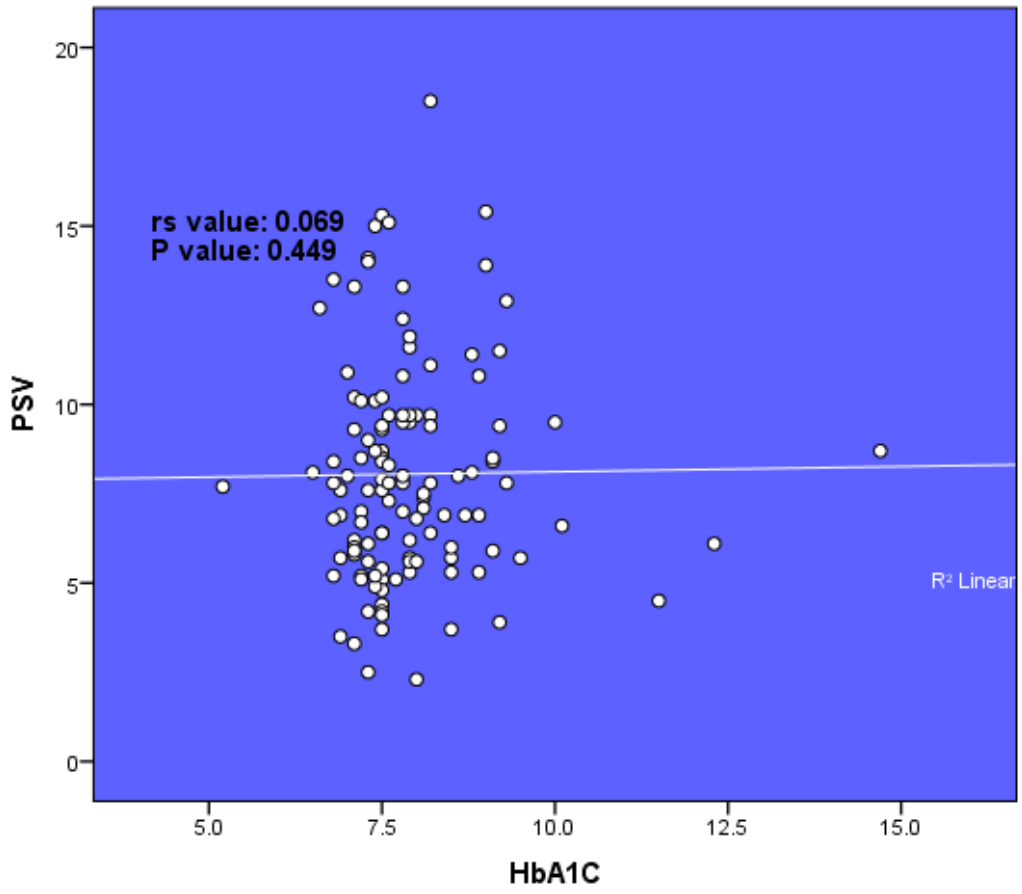


There was a weak positive correlation between HbA1c and RI (r_s value: 0.107, P value 0.238)

DOPPLER PARAMETER OF CENTRAL RETINAL VEIN:

Table 7.20: correlation between HbA1c and Doppler parameters of central retinal vein PSV among diabetic population (N=123)

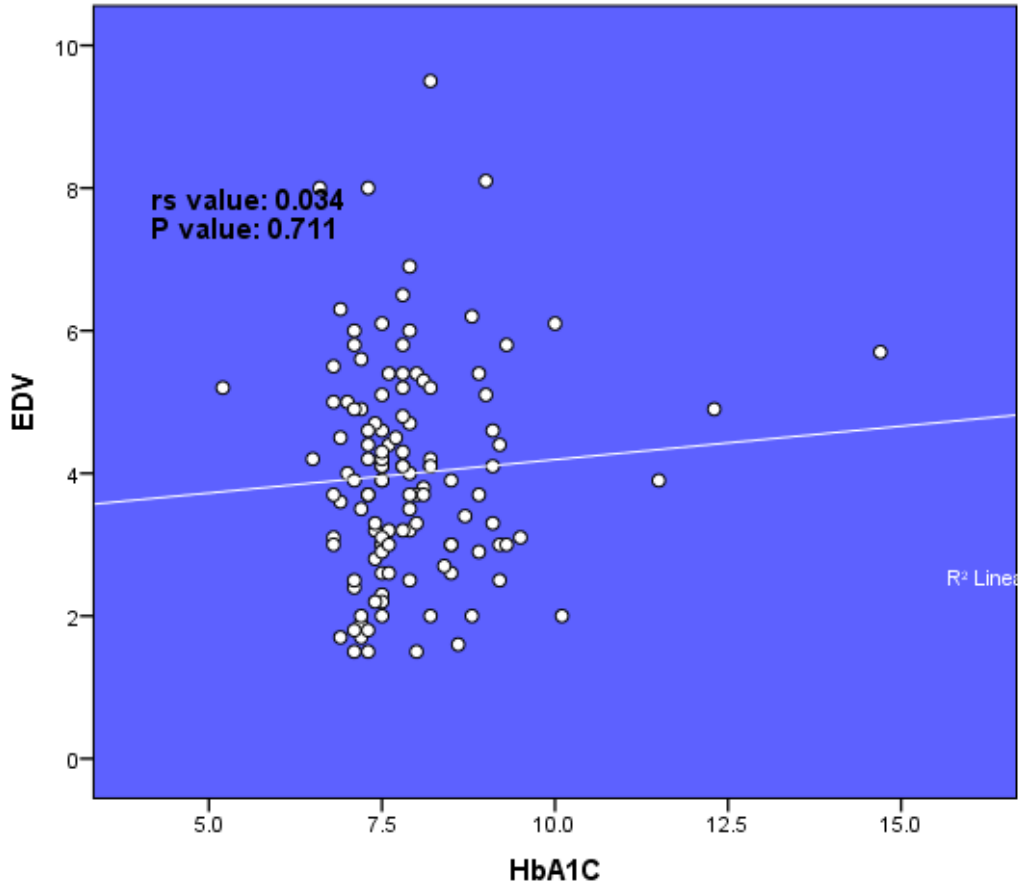
Doppler parameter of Central retinal vein	Spearman rank Correlation (r_s)	P value
PSV	0.069	0.449



There was a weak positive correlation between HbA1c and PSV (r_s value: 0.069, P value 0.449)

Table 7.21: correlation between HbA1c and Doppler parameters of central retinal vein EDV among diabetic population (N=123)

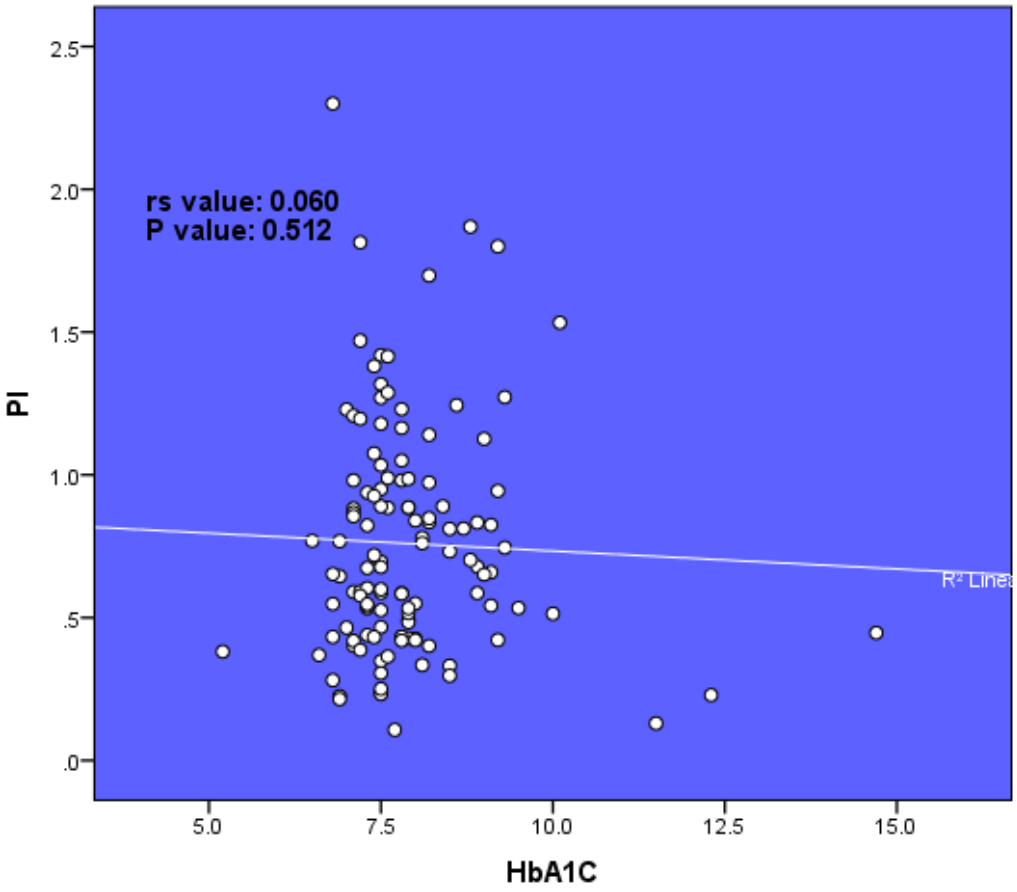
Doppler parameter of Central retinal vein	Spearman rank Correlation (r_s)	P value
EDV	0.034	0.711



There was a weak positive correlation between HbA1c and EDV (r_s value: 0.0.034, P value 0.711)

Table 7.22: correlation between HbA1c and doppler parameters of central retinal vein PI among diabetic population(N=123)

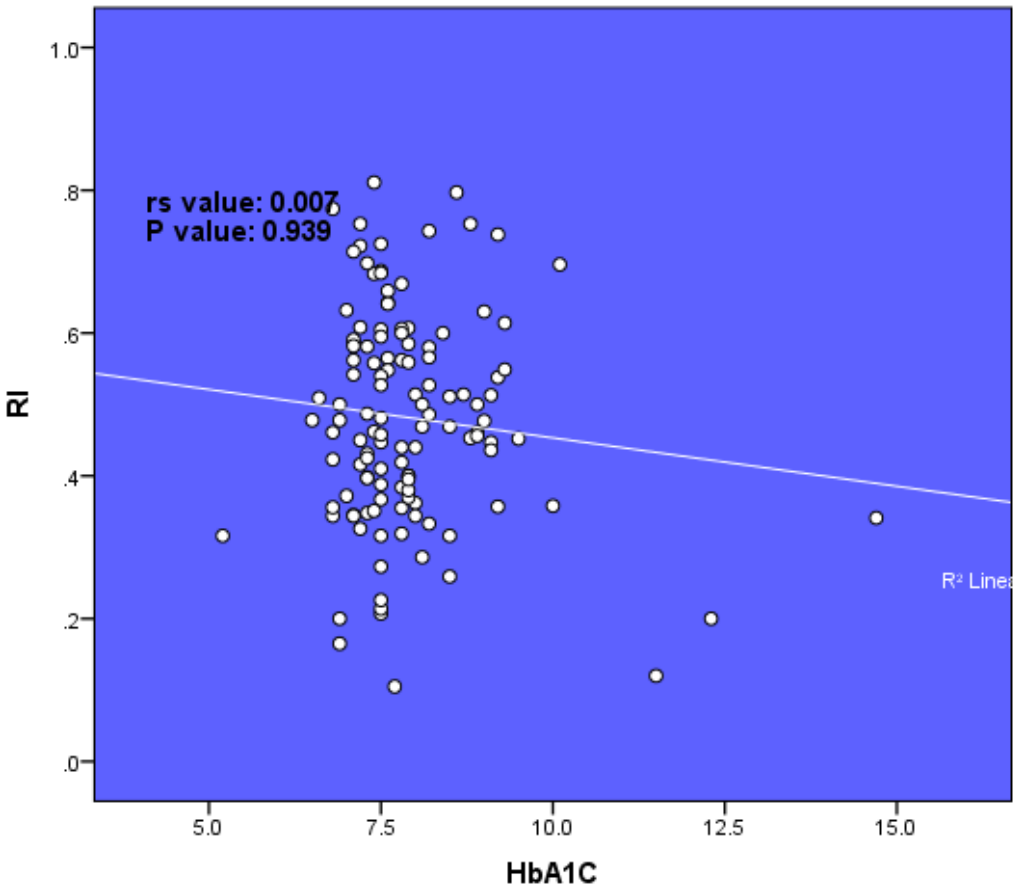
Doppler parameter of Central retinal vein	Spearman rank Correlation (r_s)	P value
PI	0.060	0.512



There was a weak positive correlation between HbA1c and PI (r_s value: 0.060, P value 0.512)

Table 7.23: Correlation between HbA1c and doppler parameters of central retinal vein RI among diabetic population(N=123)

Parameter	Spearman rank Correlation (r_s)	P value
RI	0.007	0.939



There was a weak positive correlation between HbA1c and RI (r_s value: 0.007, P value 0.939)

8. DISCUSSION

Diabetes mellitus has emerged as major public health problem in recent times, globally and in India. As per International diabetes federation estimates, in 2017 there are 451 million (age 18-99 years) diabetic population globally, which is projected to increase to 693 million by 2045[63]. As per recently published ICMR-INDIAB study, the overall prevalence of diabetes in India was 7·3% (95% CI 7·0-7·5) affecting the entire spectrum of community. This will translate into huge absolute number of patients with diabetes in India, contributing to significant chunk of diabetes population of the world [64]. Diabetes and associated metabolic derangement has adverse impact on various end organs. Intra ocular tissue, especially retinal vasculature is one of the important end organ. Diabetic retinopathy, usually reported to occur after 5 to 10 years of onset of the disease is a form of microangiopathy is the most common ocular manifestations of diabetes mellitus.[65].

Some previous studies suggested that most patients with diabetes develop characteristic abnormalities of retinal blood vessels, which include retinal capillary bed obstructions, capillary dropout, progressive thickening of the basement membrane, micro- aneurysms, venous abnormalities, arterio-venous shunts, subsequent neovasculature development, and blood rheologic abnormalities in the orbital vessels, including increased blood and plasma viscosities, increased red blood cell aggregation, increased platelet aggregation, and platelet shape changes. These abnormalities seem to disturb the retinal microcirculation and reduce the retinal blood flow if not compensated by regulatory

mechanisms. Based on the above findings, great importance should be attached to analysis of the ocular circulation in patients with diabetes[66-68].

The examinations used in assessing diabetic retinopathy usually involve imaging of the vessels in the eyeball and the retina. These include, fluorescein angiography, optical coherence tomography of the retina, B-mode ultrasound imaging, perimetry and digital retinal photography. There are many papers that discuss the correlations between retrobulbar circulation alterations and progression of diabetic retinopathy based on Doppler sonography. Color Doppler imaging is a non-invasive method enabling measurements of blood flow velocities in small vessels of the eyeball. The most frequently assessed vessels include: the ophthalmic artery, which is the first branch of the internal carotid artery, as well as the central retinal vein and artery, and the posterior ciliary arteries. The analysis of hemodynamic alterations in the retrobulbar vessels may deliver important information concerning circulation in diabetes and help to answer the question whether there is a relation between the progression of diabetic retinopathy and the changes observed in blood flow in the vessels of the eyeball. This paper presents the overview of literature regarding studies on blood flow in the vessels of the eyeball in patients with diabetic retinopathy.[3].

Color Doppler imaging has been used in ophthalmology as a safe, non-invasive method for evaluating hemodynamic alterations in the orbital vessels for 20 years.[26]This sonographic technique combines a simultaneous B-mode sonogram with colors representing movements based on Doppler frequency shifts and allows assessment of

blood flow velocities, including peak systolic velocity (PSV) and end-diastolic velocity (EDV), in retrobulbar vessels. In addition, the resistive index (RI), a measure of peripheral vascular resistance, is calculated for each retro- bulbar vessel at the same time. [40]Numerous studies have reported reduced PSV, EDV, and RI in the retro bulbar vessels, whereas others have reported increased PSV, EDV, and RI in these vessels, including the ophthalmic artery, central retinal artery, and short posterior ciliary artery, of diabetic patients without or with retinopathy.

Considering scarcity of studies comparing the retro orbital blood flow parameters using doppler among diabetic people with and without retinopathy, the current study was conducted in a tertiary care teaching hospital in south India. Among 55 people with diabetic retinopathy, 39 (70.9%) participants had mild 14 (25.5%) participants had moderate and 2(3.6%) participants had severe DR. The study also included a group of healthy controls to further understand association between diabetes and ocular blood flow parameters. The average age of all the three study groups was about 50 years, with no statistically significant difference across the study groups. In both the diabetes groups (with and without retinopathy) the proportion of males was slightly higher, as compared to equal number of males and females among control group. But the gender composition across the study groups, showed no statistically significant difference. The other key confounding variable compared across the 3 groups was systolic and diastolic blood pressure, which also hadn't shown any significant difference between diabetic population with and without retinopathy (118 mm of hg), but was slightly lesser among control

group (110 mm of Hg). The diastolic BP was 80 mm of hg in both diabetes groups, but was 72 mm of Hg among healthy group. The fasting, post prandial blood sugar values and HbA1c had shown very poor glycemic control among diabetic people with retinopathy, as compared to diabetic population without retinopathy. Among control group, the blood glucose parameters were within normal physiological range.

It is very important to consider the differences in the demographic, diabetes related parameters and co-morbidities between the study population groups, while comparing the current study findings with other studies. In a by Sood, S., et al[69]who have studied ocular blood flow parameters before and after treatment among a group of diabetics with retinopathy, before and after treatment, the age range of patients was between 38 to 75 years (mean age 55.8 ± 8.4 years). The proportion of males was 56%. These findings were comparable with diabetic population in the current study. In contrast to current study, the proportion of mild non-proliferative diabetic retinopathy (NPDR) was only 54% and higher proportion (46%) had moderate NPDR and none had severe NPDR.

COMPARISON OF OPHTHALMIC ARTERY PARAMETERS:

Among, diabetic retinopathy group, median PSV of ophthalmic artery was 32.90 (IQR 27.80 to 40.50), it was 31.25(IQR 25.92 to 34.57) among diabetic non retinopathy group, and 30.90 (IQR 29.67 to 32.52) among non-diabetic. The difference in the median PSV across study group was statistically not significant (P value 0.347). Among, diabetic retinopathy group, median EDV of ophthalmic artery was 5.20 (IQR 4.10 to 7.40), it was

6.85(IQR 5.12 to 8.47) among diabetic non retinopathy, and 7.50 (IQR 7.05 to 8.90) among non-diabetic. The difference in the median EDV across study group was statistically significant (P value <0.001).

Among, diabetic retinopathy group, median PI of ophthalmic artery was 2.04 (IQR 1.73 to 2.30), it was 1.65(IQR 1.45 to 1.87) among diabetic non-retinopathy, and 1.52 (IQR 1.36 to 1.55) among non-diabetic. The difference in the median PI across study groups was statistically significant (P value <0.001). Among, diabetic retinopathy group, median RI of ophthalmic artery was 0.83 (IQR 0.79 to 0.87), it was 0.77(IQR 0.73 to 0.80) among diabetic non-retinopathy, and 0.75 (IQR 0.72 to 0.76) among non-diabetic. The difference in the median RI across study groups was statistically significant (P value <0.001).

As per study by Sood, S., et al.[69] the PSV, EDV and RI of Ophthalmic Artery among people affected by diabetic retinopathy were 33.42 ± 18.02,7 9.12 ± 4.93 and 0.75 ± 0.07 respectively. This study had not compared the Doppler parameters with non-diabetic retinopathy/ control groups as in our study. But the Doppler parameters in diabetic people with retinopathy were closer to current study findings.

Basturk, T., et al.[70] have evaluated only resistive Index (RI) values of orbital arteries by using the color Doppler imaging (CDI) in type II diabetes mellitus (DM) patients with microalbuminuria. In this study, the DM patients with microalbuminuria were grouped into two: group 1 consisted of patients with retinopathy (n = 51) and group 2 consisted of patients without retinopathy (n = 40). Healthy subjects constituted group 3

(n = 27). Compared to diabetic group 2, group 1 had significantly higher mean RIs of OA, CRA, PCA, and HbA1c levels ($p < 0.001$ for all). Besides, there were no statistical differences in mean RIs of OA, CRA, and PCA between the control group and group 2 ($p = 1.0$; $p = 0.44$; $p = 0.67$, respectively). Mean RIs of OA and PCA were significantly correlated with age in group 1 ($r = 0.549$, $p < 0.001$; $r = 0.407$, $p = 0.003$, respectively). Mean RI of CRA was significantly correlated with the duration of diabetes and age in group 1 ($r = 0.296$, $p = 0.035$; $r = 0.486$, $p < 0.001$, respectively). The authors have concluded that, RI might be a useful marker for early diagnosis and follow-up of diabetic retinopathy, and orbital RI assessment would be beneficial for diabetic patients with retinopathy.

Similar to our study, Karami, M., et al.[71] compared orbital blood flow velocities using Doppler and gray-scale sonography in patients with DR, and to compare the results with those of their non-diabetic and diabetic peers without retinopathy. Similar to our study, compared with healthy controls, the age-adjusted resistive Index of the ophthalmic artery were significantly higher in patients with DR ($p < 0.05$). After further adjustment for age, gender, HbA1c, fasting plasma glucose, blood pressure, BMI, cholesterol, HDL, LDL, and triglycerides, only the resistivity index of the ophthalmic artery and the central retinal vein remained significantly higher in patients with DR compared with healthy controls.

Similar to our study, Krasnicki, P., et al.[57] have studied 3 groups: group I--control group, group II--type 2 diabetes patients without diabetic retinopathy, group III--

type 2 diabetes patients with non proliferative diabetic retinopathy. Peak systolic blood velocity (PSV) and end-diastolic blood velocity (ESV) in ophthalmic artery (OA) in diabetic patients were significantly lower in comparison to the control group. Peak systolic blood velocity (PSV) and end-diastolic blood velocity (ESV) in central retinal artery (CRA) were significantly lower only in patients with diabetic retinopathy. Diabetes duration was significantly longer in group III in comparison to group II. Based on the findings, the authors concluded that Blood flow in ophthalmic artery is decreased in patients with diabetes. Reduction of blood flow in central retinal artery and short posterior ciliary arteries can be significant in the development of diabetic retinopathy.

COMPARISON OF CENTRAL RETINAL ARTERY PARAMETERS:

Among, diabetic retinopathy group, median PSV of Central Retinal artery was 11.70 (IQR 7.97 to 15.45), it was 10.15(IQR 8.57 to 14.05) among diabetic without retinopathy group and 11 (IQR 10.10 to 11.85) among non-diabetic. The difference in the median PSV of Central Retinal artery across study groups was statistically not significant (P value 0.343).Among, diabetic retinopathy group, median EDV of Central Retinal artery was 2.20 (IQR 1.80 to 3.23), it was 10.15(IQR 8.57 to 14.05) among diabetic non retinopathy, and 11 (IQR 10.10 to 11.85) among non-diabetic population. The difference in the median EDV of Central Retinal artery across study groups was statistically significant (P value <0.001).Among, diabetic retinopathy group, median PI of Central Retinal artery was 1.48 (IQR 1.33 to 1.74), it was 1.46(IQR 1.30 to 1.74) among diabetic non retinopathy, and 1.35 (IQR 1.22 to 1.55) among non-diabetic. The difference in the

median PI of Central Retinal artery across study groups was statistically significant (P value <0.001). Among, diabetic retinopathy group, median RI of Central Retinal artery was 0.77 (IQR 0.79 to 0.84), it was 0.76(IQR 0.71 to 0.82) among diabetic non-retinopathy, and 0.67 (IQR 0.63 to 0.73) among non-diabetic. The difference in the median RI of Central Retinal artery across study groups was statistically significant (P value <0.001)

As per study by Sood, S., et al.[69] the PSV, EDV and RI of Central Retinal Artery among people affected by diabetic retinopathy were 14.28 ± 11.21,5.64 ± 3.81 and 0.67 ± 0.10respectively. This study had not compared the doppler parameters with non-diabetic retinopathy/ control groups as in our study. But in the diabetic population with retinopathy PSV and RI values were almost similar to current study, but EDV values appeared to be considerably lower than the current study.

Basturk, T., et al[70]have evaluated only resistive Index (RI) values of orbital arteries by using the color Doppler imaging (CDI) in type II diabetes mellitus (DM) patients with microalbuminuria. In this study, the DM patients with microalbuminuria were grouped into two: group 1 consisted of patients with retinopathy (n = 51) and group 2 consisted of patients without retinopathy (n = 40). Healthy subjects constituted group 3 (n = 27). Compared to diabetic group 2, group 1 had significantly higher mean RIs of OA, CRA, PCA, and HbA1c levels ($p < 0.001$ for all). Besides, there were no statistical differences in mean RIs of OA, CRA, and PCA between the control group and group 2 ($p = 1.0$; $p = 0.44$; $p = 0.67$, respectively). Mean RIs of OA and PCA were significantly correlated

with age in group 1 ($r = 0.549$, $p < 0.001$; $r = 0.407$, $p = 0.003$, respectively). Mean RI of CRA was significantly correlated with the duration of diabetes and age in group 1 ($r = 0.296$, $p = 0.035$; $r = 0.486$, $p < 0.001$, respectively). The authors have concluded that, RI might be a useful marker for early diagnosis and follow-up of diabetic retinopathy, and orbital RI assessment would be beneficial for diabetic patients with retinopathy.

Similar to our study, Karami, M., et al.[71] compared orbital blood flow velocities using Doppler and gray-scale sonography in patients with DR, and to compare the results with those of their non-diabetic and diabetic peers without retinopathy. Similar to our study, compared with healthy controls, the age-adjusted resistive Index of the ophthalmic artery were significantly higher in patients with DR ($p < 0.05$). After further adjustment for age, gender, HbA1c, fasting plasma glucose, blood pressure, BMI, cholesterol, HDL, LDL, and triglycerides, only the resistivity index of the ophthalmic artery and the central retinal vein remained significantly higher in patients with DR compared with healthy controls.

Similar to our study, Krasnicki, P., et al.[57] have studied 3 groups: group I--control group, group II--type 2 diabetes patients without diabetic retinopathy, group III--type 2 diabetes patients with non proliferative diabetic retinopathy. Peak systolic blood velocity (PSV) and end-diastolic blood velocity (ESV) in ophthalmic artery (OA) in diabetic patients were significantly lower in comparison to the control group. Peak systolic blood velocity (PSV) and end-diastolic blood velocity (ESV) in central retinal artery (CRA) were significantly lower only in patients with diabetic retinopathy. Diabetes duration was significantly longer in group III in comparison to group II. Based on the findings, the

authors concluded that Blood flow in ophthalmic artery is decreased in patients with diabetes. Reduction of blood flow in central retinal artery and short posterior ciliary arteries can be significant in the development of diabetic retinopathy.

Neudorfer, M., et al.[72]have assessed long-term changes in the flow parameters of retrobulbar vessels among 4 groups of eyes. Eyes of diabetic patients without diabetic retinopathy (DR),eyes with non proliferative DR, eyes with proliferative DR, and eyes of non diabetic patients (controls). The resistive index (RI) values of the central retinal artery and posterior ciliary artery had increased in the two non-DR groups and in the non proliferative DR group, with a surprising decrease measured in eyes with proliferative DR. Combining the non proliferative DR and proliferative DR groups resulted in a milder increase of the RI of the posterior ciliary artery (P= NS) and the central retinal artery (P=0.02) in the DR group compared to the other groups.

COMPARISON OF CENTRAL RETINAL VEIN PARAMETERS:

Among the, diabetic retinopathy group median PSV of central retinal vein was 8 (IQR 6 to 10.10), it was 7.60(IQR 5.60 to 9.22) among diabetic without retinopathy, and 5.50 (IQR 5 to 8.52) among non-diabetic. The difference in the median PSV of central retinal vein across study group was statistically significant (P value <0.001).

Among the diabetic retinopathy group median EDV of central retinal vein was 3.90 (IQR 2.90 to 5.20), it was 3.90(IQR 3 to 4.77) among diabetic without retinopathy, and 3.55 (IQR 3.17 to 4.32) among non-diabetic. The difference in the median EDV of central retinal vein across study group was statistically not significant (P value 0.727).

Among the, diabetic retinopathy group median PI of central retinal vein was 0.74 (IQR 0.49 to 0.98), it was 0.60(IQR 0.43 to 0.88) among diabetic without retinopathy, and 0.47 (IQR 0.38 to 0.75) among non-diabetic. The difference in the median PI of central retinal vein across study group was statistically significant (P value 0.001)

Among the, diabetic retinopathy group median RI of central retinal vein was 0.50 (IQR 0.39 to 0.60), it was 0.46(IQR 0.35 to 0.58) among diabetic without retinopathy, and 0.37 (IQR 0.30 to 0.5) among non-diabetic. The difference in the median RI of central retinal vein across study group was statistically significant (P value 0.002)

As per study by Sood, S., et al.[69] the PSV, EDV and RI of Central Retinal Vein among people affected by diabetic retinopathy were 14.28 ± 11.21,5.64 ± 3.81 and 0.67 ± 0.10 respectively. This study had not compared the doppler parameters with non-diabetic retinopathy/ control groups as in our study. But in the diabetic population with retinopathy PSV and RI values were almost similar to current study, but EDV values appeared to be considerably lower than the current study.

Dimitrova, G., et al.[53] have studied, retro bulbar circulatory parameters in type 2 diabetic patients with and without diabetic retinopathy (DR) during prospective follow up. In this study, among 18 patients who developed DR progression showed significantly increased central retinal vein PSV (5.6 (3.5-9.1) p = 0.003), EDV (3.4 (2.3-4.4) p = 0.04), and RI (0.43 (0.20-0.56) p = 0.02) at the final measurement compared to the initial measurement (PSV = 4.6 (3.2-7.0); EDV = 3.0 (2.3-3.7); RI = 0.40 (0.17-0.52)). Circulatory parameters in the central retinal artery and the posterior ciliary artery did not alter significantly after

progression of DR. 17 patients were without DR progression and they did not show any significant differences in the measured circulatory parameters on entry compared to the final measurement. The authors of this study suggest that the initial changes in the retro bulbar circulation during DR progression occur in the central retinal vein.

Basturk, T., et al. [70]have evaluated only resistive Index (RI) values of orbital arteries by using the colour Doppler imaging (CDI) in type II diabetes mellitus (DM) patients with microalbuminuria. In this study, the DM patients with microalbuminuria were grouped into two: group 1 consisted of patients with retinopathy (n = 51) and group 2 consisted of patients without retinopathy (n = 40). Healthy subjects constituted group 3 (n = 27). Compared to diabetic group 2, group 1 had significantly higher mean RIs of OA, CRA, PCA, and HbA1c levels ($p < 0.001$ for all). Besides, there were no statistical differences in mean RIs of OA, CRA, and PCA between the control group and group 2 ($p = 1.0$; $p = 0.44$; $p = 0.67$, respectively). Mean RIs of OA and PCA were significantly correlated with age in group 1 ($r = 0.549$, $p < 0.001$; $r = 0.407$, $p = 0.003$, respectively). Mean RI of CRA was significantly correlated with the duration of diabetes and age in group 1 ($r = 0.296$, $p = 0.035$; $r = 0.486$, $p < 0.001$, respectively). The authors have concluded that, RI might be a useful marker for early diagnosis and follow-up of diabetic retinopathy, and orbital RI assessment would be beneficial for diabetic patients with retinopathy.

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were significantly higher in patients with DR ($p < 0.05$). After further adjustment for age, gender, HbA1c, fasting plasma glucose, blood pressure, BMI, cholesterol, HDL, LDL, and triglycerides, only the resistivity index of the ophthalmic artery and the central retinal vein remained significantly higher in patients with DR compared with healthy controls.

Similar to our study, Krasnicki, P., et al.[57] have studied 3 groups: group I--control group, group II--type 2 diabetes patients without diabetic retinopathy, group III--type 2 diabetes patients with non proliferative diabetic retinopathy. Peak systolic blood velocity (PSV) and end-diastolic blood velocity (ESV) in ophthalmic artery (OA) in diabetic patients were significantly lower in comparison to the control group. Peak systolic blood velocity (PSV) and end-diastolic blood velocity (ESV) in central retinal artery (CRA) were significantly lower only in patients with diabetic retinopathy. Diabetes duration was significantly longer in group III in comparison to group II. Based on the findings, the authors concluded that Blood flow in ophthalmic artery is decreased in patients with diabetes. Reduction of blood flow in central retinal artery and short posterior ciliary arteries can be significant in the development of diabetic retinopathy.

CORRELATION OF HBA1C AND DOPPLER PARAMETERS: There was a weak positive correlation between HbA1c and PSV (r_s value: 0.048, P value 0.601). There was a weak negative correlation between HbA1c and EDV (r_s value: -0.132, P value 0.145). There was a weak positive correlation between HbA1c and PI (r_s value: 0.204, P value 0.024). There was a weak positive correlation between HbA1c and RI (r_s value: 0.147, P value 0.106). There was a weak negative correlation between HbA1c and PSV (r_s value: -

0.025, P value 0.781). There was a weak negative correlation between HbA1c and EDV (r_s value: -0.115, P value 0.208). There was a weak positive correlation between HbA1c and PI (r_s value: 0.055, P value 0.547). There was a weak positive correlation between HbA1c and RI (r_s value: 0.107, P value 0.238).There was a weak positive correlation between HbA1c and PSV (r_s value: 0.069, P value 0.449).There was a weak positive correlation between HbA1c and EDV (r_s value: 0.0.034, P value 0.711). There was a weak positive correlation between HbA1c and PI (r_s value: 0.060, P value 0.512).There was a weak positive correlation between HbA1c and RI (r_s value: 0.007, P value 0.939)

Sood, S., et al.[69] concluded that, alterations in the retro bulbar arterial circulation do not seem to predict the progression of diabetic retinopathy and development of proliferative disease in NIDDM patients. The retro bulbar arterial circulation appeared to be affected in all diabetics and the changes appear to progress with increasing duration in all diabetics. Certain other factors seem to play a role in progression of DR.

Diabetes duration was the parameter, which had consistently demonstrated a strong association with the changes in orbital blood flow parameters in many previous studies. Basturk, T., et al.[70] have reported Mean RI of CRA to be significantly correlated with the duration of diabetes and age in patients with DR ($r = 0.296$, $p = 0.035$; $r = 0.486$, $p < 0.001$, respectively). Study by Krasnicki, P., et al.[57] also have reported similar findings. This relationship has not been evaluated by our study.

As in our study, Meng, N., et al.[73] have done Color Doppler Imaging Analysis of Retrobulbar Blood Flow Velocities in 3 groups: group 1, diabetes without retinopathy

versus control; group 2, DR versus control; and group 3, diabetic without retinopathy versus DR. In group 1, eyes without retinopathy had a significant increase in ophthalmic artery PSV (P = .002) and significant reductions in central retinal artery PSV and EDV (P = .002; P = .007, respectively). A significant increase in ophthalmic artery RI (P = .02) was found in eyes without retinopathy. In group 2, central retinal artery PSV and EDV in eyes with retinopathy decreased significantly (P < 0.00001). Ophthalmic artery RI was significantly higher in eyes with retinopathy than controls (P = .0008). In group 3, ophthalmic artery PSV was lower in eyes with retinopathy (P= .04) than eyes without, and central retinal artery PSV and EDV decreased significantly (P = .004; P < .00001) in eyes with retinopathy compared to eyes without. Differences in ophthalmic and central retinal artery RIs were also found in eyes with retinopathy (P = .05; P < .00001). In one of the early studies, Mendivil, A., et al.[47] have compared blood flow velocity in ocular vessels (ophthalmic artery, posterior ciliary arteries, central retinal vessels, and vortex veins) of 25 patients with proliferative diabetic retinopathy and 30 age matched normal subjects using a colour Doppler imaging unit. The diabetic patients had lower blood velocities than the volunteers. There were significant differences in ophthalmic artery; systolic ($p < 0.01$), diastolic ($p < 0.001$), mean velocity ($p < 0.05$), and central retinal artery; systolic ($p < 0.001$), diastolic ($p < 0.001$), mean velocity ($p < 0.05$). No significant correlations were found between age and blood velocities.

Ocular blood flow velocity was decreased in diabetic patients with retinopathy with increased PI and RI.

9. LIMITATIONS OF THE STUDY

However our study had some limitations which are listed below

1. Lack of statistical significance of differences in many quantitative parameters across the study groups may be attributed to relatively smaller sample of the study.
2. The role of positive or negative confounding effect by various demographic and clinical variables could not be tested due to smaller sample size
3. Generalization of the study findings was limited, as the study was conducted in a single centre with limited catchment area
4. There are many different results relating to the association of blood flow velocities and diabetic retinopathy in the literature.
5. The interpretation of these data is complicated by the demographic variability in the patients, the methodological problems related to the outcome measures in these studies, and their subsequent analyses.
6. Colour doppler Imaging is operator-dependent nature of ultrasonography although it is non-invasive, easily performed, and confidential method for evaluating CDI

10. SUMMARY

- The current study had compared the ocular blood flow parameters, among diabetic patients, with and without retinopathy and non-diabetic healthy control group.
- The average age of all the three study groups was about 50 years, with no statistically significant difference across the study groups.
- In both the diabetes groups (with and without retinopathy) the proportion of males was slightly higher, as compared to equal number of males and females among control group. But the gender composition across the study groups, showed no statistically significant difference.
- Among the people with diabetic retinopathy, majority (70.9%) participants had mild DR. The proportion of moderate and severe DR was 25.5% and 3.6% respectively.
- Even though the systolic blood pressure was slightly higher and showed statistically significant difference in healthy controls, as compared to other two groups, the difference doesn't appear to be of any clinical significance. No clinically significant difference was found in diastolic blood pressure across three groups.
- All the glycemic control parameters (fasting blood sugar, post prandial blood sugar and HbA1c) were significantly higher in diabetes with retinopathy group, as compared to diabetes without retinopathy, which had comparatively higher values than normal healthy controls.

- Among, **ophthalmic artery** doppler parameters, PSV had shown no statistically significant difference across the groups (P value 0.347). EDV was lowest in DR group, followed by non-DR group and was higher in healthy controls (P value <0.001). The PI was highest in DR group, followed by no DR group and least among healthy controls (P value <0.001). The RI of **ophthalmic artery was also highest among DR group and showed declining trend in non DR and healthy controls** (P value <0.001)
- Among Central Retinal artery parameters also PSV did not show any statistically significant difference. EDV of Central Retinal artery was much lower in DR group, as compared to non-DR group and healthy controls. PI and RI of **Central Retinal artery** also showed declining trend from DR group to non-DR group and healthy controls (P value <0.001).
- Among the, **central retinal vein** parameters PSV was highest in DR group and showed declining trend in non-DR group and (healthy controls. (P value <0.001). EDV of both DR and non-DR groups was similar and even though, it was slightly lower in control group, the difference was statistically not significant P value 0.727). The PI and RI values of **central retinal vein** were highest in DR group, followed by non DR group and lowest in healthy controls (P value <0.05)
- None of the ocular blood flow parameters had shown statistically significant correlation with HbA1c values among diabetic population.

11. CONCLUSION

1. This study concludes that Orbital Colour Doppler Imaging has the potential to provide useful information related to altered ocular blood flow even before the appearance of Diabetic retinopathy thereby enabling early diagnosis of diabetic retinopathy and early intervention.
2. The findings of our study suggest a need for large-scale studies to derive a cut off value in the Doppler indices to identify the Diabetics who are at risk of developing retinopathy. Further the study of association of ocular blood flow parameters with other micro and macro vascular complications in diabetics may help in identifying subjects with high risk of developing complications.
3. This study also opens up the area of research required in assessment of treatment response with the utility of colour Doppler imaging in Diabetic Retinopathy.

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ABBREVIATIONS

USG	-Ultrasonogram
CDI	-Colour Doppler Imaging
DM	-Diabetes Mellitus
DR	-Diabetic Retinopathy
PDR	-Proliferative Diabetic Retinopathy
NPDR	-Non Proliferative Diabetic Retinopathy
PSV	-Peak Systolic Velocity
EDV	-End Diastolic Velocity
PI	-Pulsatility Index
RI	-Resistivity Index
OA	-Opthalmic Artery
CRA	-Central Retinal Artery
PCA	-Posterior Ciliary Artery
CRV	-Central Retinal Vein
ADA	- American Diabetes Association
WHO	- World Health

PROFORMA

STUDY TITLE:

"COMPARATIVE STUDY OF ORBITAL DOPPLER PARAMETERS IN DIABETICS WITH RETINOPATHY AND DIABETICS/ HEALTHY CONTROLS WITHOUT RETINOPATHY"

SI.NO:

NAME:

AGE/SEX:

OCCUPATION:

ADDRESS:

PRESENTING COMPLAINTS:

PAST HISTORY:

HYPERTENSION

DIABETES MELLITUS

EXAMINATION: BLOOD PRESSURE MEASUREMENT

INVESTIGATIONS:

FASTING BLOOD SUGAR

POST PRANDIAL BLOOD SUGAR

FUNDOSCOPY

ORBITAL DOPPLER IMAGING FINDINGS:

VARIABLE	DIABETICS WITH RETINOPATHY	DIABETICS WITHOUT RETINOPATHY AND HEALTHY CONTROLS
OPTHALMIC ARTERY PSV(cm/sec) EDV(cm/sec) RI PI		
CENTRAL RETINAL ARTERY PSV(cm/sec) EDV(cm/sec) RI PI		
CENTRAL RETINAL VEIN PSV(cm/sec) EDV(cm/sec) RI PI		

INTERPRETATION:

PATIENT INFORMATION SHEET

We are conducting a study on "COMPARATIVE STUDY OF ORBITAL DOPPLER PARAMETERS IN DIABETICS WITH RETINOPATHY AND DIABETICS/ HEALTHY CONTROLS WITHOUT RETINOPATHY"

Your cooperation would be valuable for the same

The privacy of patients in the research will be maintained throughout the study.In the event of any publication or presentation resulting from the research, no personally identifiable information will be shared.

Taking part of the study is voluntary.you are free to decide whether to participate in this study or to withdraw at any time.your decision will not result in any loss of benefits to which your are otherwise entitled.

The result of the special study may be intimated to you at the end of the study period or during the study if anything is found abnormal which may aid in the management or treatment.

Signature of the investigator Signature of the participant

Date:

ஆராய்ச்சி தகவல் தாள்

நீரிழிவு விழித்திரை நோய் உள்ளவர்களுக்கும் மற்றும் இல்லாதவர்களுக்கும் கண்களுக்கான டாப்ளர் அல்ட்ராசவுண்ட் குறியீடுகளின் அளவுகளை ஒப்பிடும் ஆய்வு

இந்த ஆய்வு மூலம் நீரிழிவு நோயினால் ஏற்படும் விழித்திரை நோயினை முன் கூட்டியே அறிய உதவும் என்பதை தெரிவித்து கொள்கிறோம்.

இந்த ஆய்வில் செய்யப்பட உள்ளகண்களுக்கான டாப்லர் அல்ட்ராசவுண்ட் மற்றும் ∴பண்டாஸ்கோபி பரிசோதனையில் எந்த பக்க விளைவுகளும் ஏற்படாது.

இந்த அராய்ச்சியில் பங்கேற்பது தங்களுடைய விருபத்தின் பேரில் தான் இருக்கிறது. மேலும் நீங்கள் எந்நேரமும் இந்த ஆராய்ச்சியிலிருந்து பின்வாங்கலாம் என்பதையும் தெரிவித்துக் கொள்கிறோம்.

இந்த சிறப்பு சிகிச்சையின் முடிவுகளை ஆராய்ச்சியின் போது அல்லது ஆராய்ச்சியின் முடிவின் போது தங்களுக்கு அறிவிக்கப்படும் என்பதையும் தெரிவித்து கொள்கிறோம்.

ஆராய்ச்சியாளர் கையொப்பம் பங்கேற்பாளர் கையொப்பம்

நாள் :

இடம் :

PATIENT CONSENT FORM

STUDY TITLE : "COMPARATIVE STUDY OF ORBITAL DOPPLER PARAMETERS IN DIABETICS WITH RETINOPATHY AND DIABETICS/ HEALTHY CONTROLS WITHOUT RETINOPATHY"

PARTICIPANT

Name: Age: Sex: IP NO.

I confirm that I have understood the purposes of procedure for the above study.I have the opportunity to ask the questions and all my questions and doubts have been answered to my satisfaction. ☐

I have been explained about the pitfall in the procedure. I have been explained about the safety, advantage and disadvantage of the technique. I understand that my participation in the study is voluntary and that i am free to withdraw at anytime without giving any reason. ☐

I understand that investigator, regulatory authorities and the ethics committee will not need my permission to look at my health records both in respect to current study and any further research that may be conducted in relation to it, even if I withdraw from the study. ☐

I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from the study. ☐

I have been explained that the ORBITAL DOPPLER IMAGING STUDY technique is a standard and approved technique. This may help in future research in the field of radiology. I consent to undergo this procedure. ☐

I HEREBY CONSENT TO UNDERGO COMPLETE PHYSICAL EXAMINATION, BIOCHEMICAL AND RADIOLOGICAL INVESTIGATION PERTAINING TO THE STUDY.

DATE: SIGNATURE/THUMB IMPRESSION OF PATIENT

ஆராய்ச்சி ஒப்புதல் கடிதம்

ஆராய்ச்சி தலைப்பு

நீரிழிவு விழித் திரை நோய் உள்ளவர்களுக்கும் மற்றும் இல்லாதவர்களுக்கும் கண்களுக்கான டாப்ளர் அல்ட்ராசவுண்ட் குறியீடுகளின் அளவுகளை ஒப்பிடும் ஆய்வு

பெயர் : தேதி :

வயது உள்நோயாளி எண் :

பால் ஆராய்ச்சிசேர்க்கை எண் :

இந்த ஆராய்ச்சியின் விவரங்களும் அதன் நோக்கங்களும் முழுமையாக எனக்கு தெளிவாக விளக்கப்பட்டது.

எனக்கு விளக்கப்பட்ட விஷயங்களை நான் புரிந்து கொண்டு எனது சம்மதத்தை தெரிவிக்கிறேன்.

இந்த அராய்ச்சியில் பிறரின் நிர்பந்தமின்றி என் சொந்த விருப்பதின் பேரில் பங்கு பெறுகின்றேன்.

இந்த ஆரய்ச்சியில் இருந்து நான் எந்நேரமும் பின் வாங்கலாம் என்பதையும் அதனால் எந்த பாதிப்பும் எற்படாது என்பதையும் நான் புரிந்து கொண்டேன்.

நான் இந்த ஆராய்ச்சியின் விபரங்களைக் கொண்ட ஆராய்ச்சித் தகவல் தாளைப் பெற்றுக்கொண்டேன்.

இதன் மூலம் எந்த பின் விளைவும் எற்படாது என்று மருத்துவர் மூலம் தெரிந்து கொண்டு, நான் என்னுடைய சுயநினைவுடனும் மற்றும் முழுசுதந்திரத்துடனும் இந்த மருத்துவ அராய்ச்சியில் என்னை சேர்த்துக்கொள்ள சம்மதம் தெரிவிக்கிறேன்.

தேதி : பங்கேற்பாளர் கையொப்பம்

S. No.	Group	NAME	AGE	SEX	Height	Weight	SBP	DBP	FBS	PPBS	HbA1C	OPHTHALMIC ARTERY	OAEDV	OAPI	OARI	CENTRAL RETINAL ARTERY				CENTRAL RETINAL VEIN				FUNDOSCOPY
												OAPSV				CRAPSV	CRAEDV	CRAPI	CRARI	CRVPSV	CRVEDV	CRVPI	CRVRI	
1	ND	CHELLAMMAL	65	F	166	78	110	80	78	128	5.6	32.3	6.6	1.62	0.797	7.8	2	1.23	0.743	4	1	1.48	0.75	normal
2	ND	BANU	40	F	148	56	110	80	96	118	5.1	31	10	1.2	0.68	4.01	1.87	0.87	0.53	3.48	1.01	1.176	0.709	NORMAL
3	ND	RAJESHWARI	50	F	161	60	110	70	80	102	5.1	63.6	22.4	0.999	0.648	13.3	4.4	1.135	0.676	14.4	5.4	0.947	0.622	normal
4	ND	AMULU	60	F	155	60	108	72	89	128	4.9	20	5	1.299	0.753	8.3	1.6	2.2	0.804	9.3	3.4	1.32	0.633	normal
5	ND	VEERAMMAL	60	F	158	56	110	80	26.9	6	4.1	26.9	6	1.662	0.776	6	2.8	0.928	0.595	5.6	1.8	1.397	0.681	normal
6	ND	BALAJI	65	M	160	65	100	70	160	65	4.9	54.1	14.8	1.373	0.726	11.3	3.7	1.095	0.671	6.5	4	0.417	0.375	normal
7	ND	RAVIRAJ	50	M	165	65	118	72	88	110	5.3	23.7	6	1.53	0.748	11.2	2.2	1.605	0.8	11.1	4.4	0.996	0.605	normal
8	ND	RASAMMAL	60	f	168	72	120	80	86	112	4.9	44.4	12.3	1.5	0.724	19.8	5.8	1.146	0.709	5.6	2.6	0.931	0.531	NORMAL
9	ND	VASANTHI	50	F	156	60	110	80	80	122	5.1	46.4	6.5	1.849	0.869	11.1	2.9	1.507	0.741	9.6	4.1	0.96	0.574	normal
10	ND	MARIMUTHU	60	M	163	59	120	70	88	124	5.4	37.7	18.4	0.73	0.512	15	5.3	1.065	0.64	5.5	2.5	0.75	0.545	normal
11	ND	SELVI	57	F	164	71	108	78	76	111	5.1	26.9	6.5	1.47	0.758	16	5.5	1.08	0.659	6.4	4.9	0.66	0.531	normal
12	ND	ANBU	26	M	162	58	122	78	78	108	5.3	26.9	6.5	1.474	0.758	14.4	5.9	0.949	0.589	8	4.4	0.605	0.589	normal
13	ND	JAYALAKSHMI	48	F	164	68	110	80	85	102	5.2	32.2	8.5	1.7	0.36	11.8	2.1	1.3	0.82	8.3	4.1	0.45	0.506	normal
14	ND	MURUGESH	60	M	163	68	118	72	102	120	5	49.8	10.6	1.547	0.788	7.7	1.8	1.543	0.769	6.2	2.3	1.064	0.63	normal
15	ND	RAJESH	50	M	170	80	120	70	78	118	4.6	23.4	6	1.6	0.743	13.2	3.2	1.348	0.754	6.3	2.5	1.079	0.593	normal
16	ND	VELU	62	M	165	60	110	70	90	129	4.9	17.8	4.1	1.493	0.767	13	2.9	1.515	0.778	11.3	3.5	1.546	0.688	NORMAL
17	ND	PARVATHY	56	F	165	70	110	78	92	118	5.2	21.2	5.1	1.663	0.758	7.3	2.5	1.051	0.658	5.8	2.8	0.45	0.517	normal
18	ND	PUSHPA	35	F	163	60	118	82	102	125	5.2	34.9	9.7	1.27	0.621	13	4.7	1.322	0.637	11.6	5.4	0.631	0.533	NORMAL
19	ND	ANJALAI	28	F	159	60	110	70	98	122	5.1	28.4	8.9	1.3	0.688	6.9	2.6	0.926	0.618	7.4	5.7	0.41	0.229	normal
20	ND	RAJESHWARI	29	F	164	68	110	80	101	128	5.2	34.9	7.8	1.595	0.776	7.3	2.5	1.051	0.658	6.9	2.9	0.982	0.571	normal
21	ND	VANMATHI	33	F	164	60	120	80	108	128	5.3	22.3	7.1	1.235	0.68	10.8	2.3	1.59	0.786	5.2	2.9	0.48	0.44	normal
22	ND	ELUMALAI	60	M	160	50	110	80	84	120	5.1	41.5	9.4	1.49	0.773	17.1	7	0.953	0.592	12.2	5.1	1.008	0.583	normal
23	ND	BHUVANESHWARI	27	f	160	50	110	60	80	112	5.1	44.5	16.1	1	0.638	11.5	3.2	1.115	0.724	12.4	5.4	1.036	0.566	NORMAL
24	ND	REVATHY	37	F	156	62	110	70	81	128	4.9	36.8	14.5	0.838	0.607	10.9	5.4	0.614	0.504	10.2	3.8	1.259	0.632	normal
25	ND	SUBRAMANI	56	M	165	65	122	78	98	128	4.8	32.5	10.5	1.106	0.677	11.3	4.2	1.024	0.632	8.7	4.1	0.875	0.534	normal
26	ND	VINOTHKUMAR	35	M	159	72	100	82	80	121	5.1	36.9	9.7	1.7	0.737	13.3	7.5	0.564	0.44	6.4	3.6	0.604	0.432	normal
27	ND	SATHISH	30	M	168	60	126	80	110	70	4.9	31.6	10.01	1.082	0.684	12	5.9	0.747	0.504	12.7	8	0.509	0.369	normal
28	ND	pandiyan	59	M	155	52	120	80	81	123	4.9	28	11.6	0.951	0.585	9.9	3.1	1.208	0.686	6	3.7	0.489	0.387	normal
29	ND	PANDU	49	M	159	50	112	78	78	112	4.5	23	9.7	0.933	0.581	11.5	6.7	0.531	0.42	9	6	0.425	0.333	normal
30	ND	SELVARAJ	45	m	168	65	110	78	85	123	5.2	22.7	7.3	1.254	0.677	19.8	6.7	1.3	0.66	9.1	6	0.418	0.34	normal
31	ND	SUBRAMANI	49	M	165	64	112	80	84	124	5.5	23.8	5.8	1.556	0.754	21.7	6	1.299	0.724	8.6	6	0.367	0.298	normal
32	ND	PRAKASAM	63	M	168	64	110	80	89	119	5.6	38	9	1.722	0.764	10.2	2.6	1.443	0.74	6.7	3.1	0.921	0.545	normal
33	ND	PRABHAKARAN	60	M	170	69	112	80	92	128	5.1	33.6	6.7	1.959	0.8	11.5	2.1	1.737	0.815	4.4	2.5	0.572	0.44	normal
34	ND	MUNUSAMY	50	M	164	70	120	80	79	124	5.6	37.1	12.2	1.188	0.671	9.1	2.2	1.318	0.755	4.9	3.2	0.408	0.346	normal
35	ND	MUNIYAN	55	M	166	68	110	78	85	128	5.4	28.3	7.1	1.451	0.748	9.4	1.9	1.415	0.8	11.1	4.3	1.217	0.611	normal
36	ND	GANAPATHY	52	M	160	62	120	62	83	110	5.2	30.1	6.4	1.771	0.786	17	3.4	1.559	0.798	7.7	5.2	0.381	0.316	normal
37	ND	GOPAL	56	M	162	65	120	80	78	120	5.5	33.1	6.1	1.985	0.816	12	2.5	1.443	0.789	5.9	4.2	0.331	0.286	normal
38	ND	PANDURANGAN	55	M	156	48	110	60	85	128	5	36	11.7	1.178	0.676	15.1	3.6	1.749	0.763	8.8	5.5	0.525	0.378	normal
39	ND	PARAMASIVAN	50	M	165	52	120	80	80	131	5.1	31.9	6.5	1.664	0.795	8.4	3.5	0.91	0.581	14.9	6.7	0.82	0.545	normal
40	ND	PORKODI	53	F	165	62	120	70	98	108	5.2	20.5	7.3	1.082	0.643	5.5	2.2	0.825	0.6	5.5	2.2	0.308	0.267	normal
41	ND	LATHA	50	F	162	65	110	72	89	108	4.9	34.7	12.7	1.085	0.635	4.3	1.5	0.937	0.641	4.8	2.1	0.704	0.557	normal
42	ND	THILAGAVATHY	55	F	165	72	110	60	101	128	4.2	30.9	8.3	1.484	0.731	13.9	6.1	0.915	0.561	10.4	4.3	1.055	0.585	normal
43	ND	THENMOZHI	40	F	170	62	110	70	99	108	4.1	30	7.7	1.523	0.745	9.4	4.2	0.796	0.547	4.8	4.2	0.116	0.111	normal
44	ND	ANGEL	30	F	166	62	110	70	89	106	4.5	49.6	8.9	1.075	0.82	13.9	4	1.312	0.716	8.5	5.6	0.422	0.341	normal
45	ND	AMULU	40	F	164	58	110	80	80	120	4.6	18.7	4	1.366	0.784	25	5.1	1.538	0.794	11	6.8	0.475	0.383	normal
46	ND	AMALA	60	F	168	59	120	80	102	128	4.6	24.3	6.5	1.297	0.731	17.8	4	1.611	0.776	8.2	4.9	0.523	0.4	normal

S. No.	Group	NAME	AGE	SEX	Height	Weight	SBP	DBP	FBS	PPBS	HbA1C	OPHTHALMIC ARTERY	OAEDV	OAPI	OARI	CENTRAL RETINAL ARTERY				CENTRAL RETINAL VEIN				FUNDOSCOPY
												OAPSV				CRAPSV	CRAEDV	CRAPI	CRARI	CRVPSV	CRVEDV	CRVPI	CRVRI	
47	ND	MOHAMMMED BASHA	45	M	166	54	110	80	101	130	4.8	32.3	5.9	1.713	0.817	9.6	2.3	1.363	0.761	8.8	5.1	0.587	0.423	normal
48	ND	BASHEERA	47	F	162	55	110	60	92	118	4.9	29.4	8	1.353	0.727	10.9	1.6	1.854	0.853	6	2.2	0.927	0.638	normal
49	ND	EZHUMALAI	56	M	160	49	110	70	98	133	4.8	35.5	8.9	1.667	0.749	9.5	2.8	1.274	0.705	4.7	2.8	0.498	0.4	normal
50	ND	ELIZABETH	50	F	149	50	110	70	90	113	4.2	30.4	9.2	1.37	0.696	7.7	1.4	1.559	0.823	5.4	4.3	0.221	0.2	normal
51	ND	ALFRINA	23	F	148	52	110	78	102	128	4.8	30.1	7.5	1.503	0.75	11.6	3.1	1.433	0.735	9.3	5.5	0.562	0.623	normal
52	ND	DHANAPAL	39	M	152	60	120	78	108	123	5.1	28.9	6.9	1.546	0.761	14.8	5.2	1.14	0.851	7.2	5.5	0.775	0.653	normal
53	ND	DEVARAJAN	60	M	148	52	110	60	82	118	4.9	32.1	8.2	1.478	0.744	12.7	6.5	0.732	0.486	9.5	5.7	0.545	0.611	normal
54	ND	NAGENDRAN	56	M	152	60	110	70	88	120	4.4	31.8	10.5	1.21	0.67	10.5	3.8	1.11	0.638	5.5	3.2	0.58	0.418	normal
55	ND	NOORJAHAN	45	F	159	64	110	80	92	128	4.5	28.4	7.5	1.445	0.735	11.8	3.8	1.238	0.677	4.8	3.5	0.33	0.27	normal
56	ND	NARASIMMAN	50	M	160	65	110	70	98	132	4.9	31.1	7.6	1.5	0.755	10.3	3.8	1.09	0.631	5.1	3.5	0.397	0.313	normal
57	ND	VIDHYA	45	F	160	49	110	60	108	124	4.7	29.8	7.5	1.496	0.748	9.9	3.8	1.04	0.616	5.2	3.5	0.418	0.326	normal
58	ND	VARADHAN	55	M	155	52	110	70	118	128	4.5	30.2	7.5	1.507	0.751	11.1	3.5	1.26	0.684	5.1	3.2	0.496	0.171	normal
59	ND	MAHALAKSHMI	40	F	149	52	110	60	108	125	4.5	31	6.8	1.628	0.78	10.9	3.8	1.151	0.651	5.1	3.9	0.279	0.235	normal
60	ND	MEERAN	45	M	155	55	110	70	110	138	4.8	29.7	7.2	1.53	0.757	10.8	2.9	1.482	0.731	4.8	3.2	0.428	0.333	normal
61	ND	VENDA	60	F	162	59	118	72	98	123	4.5	29.8	7.6	1.48	0.744	10.9	3.9	1.123	0.642	4.9	3.5	0.353	0.285	normal
62	ND	MEENA	40	F	165	55	120	80	88	113	4.2	31.2	7.6	1.525	0.756	10.5	3.2	1.295	0.695	5.2	2.8	0.666	0.461	normal
63	ND	MEERA	55	F	159	62	110	80	92	113	4.1	29.7	7.2	1.53	0.762	10.1	3.6	1.127	0.643	4.9	3.6	0.322	0.17	normal
64	ND	MUTHU	60	M	160	60	110	70	88	133	4.9	30.1	7.1	1.558	0.764	10.9	3.2	1.336	0.706	4.7	3.9	0.192	0.17	normal
65	ND	RAJESH	55	M	155	62	120	80	92	131	4.5	31.2	7.3	1.565	0.766	9.9	4.1	0.961	0.585	4.3	3.1	0.342	0.279	NORMAL
66	ND	SANKARAN	45	M	168	68	120	80	98	124	5.2	29.6	7.2	1.527	0.756	10.5	3.8	1.11	0.538	5.1	3.4	0.428	0.333	normal
67	ND	RADHA	49	F	162	42	110	60	88	116	4.1	30.4	7.3	1.54	0.759	10.9	3.5	1.24	0.678	5.1	3.8	0.307	0.254	normal
68	ND	MARY	50	F	158	52	110	70	90	125	4.9	30.1	7.1	1.558	0.764	10.8	3.2	1.32	0.703	4.5	2.9	0.47	0.355	normal
69	ND	MAHISHA	50	F	165	40	110	80	78	109	4.2	31.8	8.1	1.46	0.745	11.1	3.6	1.229	0.675	4.8	3.6	0.3	0.25	normal
70	ND	KANDHAN	60	M	164	58	110	70	92	118	4.3	31.3	7.5	1.54	0.76	11.1	3.8	1.171	0.657	5.1	3.5	0.396	0.313	normal
71	ND	VELAYUTHAM	50	M	163	59	110	80	89	107	4.2	30.8	7.2	1.54	0.766	9.9	2.8	1.22	0.717	5.1	3.5	0.41	0.313	normal
72	ND	VEERA	45	M	165	52	110	70	92	117	4.3	30.4	7.1	1.567	0.766	10.9	3.5	1.24	0.678	4.9	2.9	0.56	0.408	normal
73	ND	MARIYAMMAL	50	F	162	52	110	80	98	124	4.4	29.9	7.2	1.537	0.759	11.1	3.2	1.35	0.711	5.3	3.7	0.377	0.301	normal
74	ND	SAROJA	50	F	166	54	110	60	101	128	5.2	30.3	7.5	1.509	0.752	11.3	3.5	1.27	0.69	4.8	3.5	0.33	0.27	normal
75	ND	LATHA	46	F	168	59	110	70	99	128	4.5	29.7	7.1	1.54	0.76	11.2	3.5	1.27	0.687	4.8	3.6	0.3	0.25	normal
76	ND	MEGHANATHAN	50	M	164	58	110	60	98	123	4.6	31.2	8.3	1.566	0.766	10.1	3.2	1.254	0.683	5.1	3.5	0.397	0.313	normal
77	ND	RAVI	45	M	167	64	120	80	102	129	4.9	30.1	7.1	1.558	0.764	10.1	2.9	1.35	0.72	5.2	3.5	0.418	0.326	normal
78	ND	RAGHAVAN	46	M	165	63	110	70	108	128	4.3	31.1	7.5	1.536	0.758	11.3	3.2	1.37	0.716	5.1	3.2	0.5	0.372	normal
79	ND	SUBHA	46	F	165	53	110	60	101	126	4.1	31.4	8.2	1.5	0.738	10.3	4.1	1.006	0.601	5.1	3.9	0.27	0.235	normal
80	ND	RAMESH	50	M	168	59	110	60	108	129	4.2	32.1	8.3	1.469	0.741	11.5	3.9	1.18	0.66	5.6	3.9	0.381	0.303	normal
81	ND	VEERAPPAN	56	M	164	60	120	80	106	123	4.5	30.5	7.7	1.49	0.747	11.1	3.2	1.362	0.711	4.9	2.5	0.727	0.489	normal
82	ND	RAJA	48	M	167	61	120	70	81	118	5	31.6	7.2	1.594	0.772	10.9	3.9	1.12	0.642	4.8	3.5	0.333	0.27	normal
83	ND	VALLI	52	F	158	62	110	70	98	110	4.9	30.2	6.9	1.595	0.771	11.3	4.5	1.005	0.681	5.2	3.6	0.387	0.307	normal
84	ND	LAKSHMI	61	F	152	55	110	60	88	115	4.5	31.2	6.8	1.637	0.782	11.5	3.5	0.568	0.695	5	3.2	0.473	0.36	normal
85	ND	NALINI	45	F	162	58	120	70	92	128	4.6	32.6	7.8	1.55	0.76	11.1	3.6	1.22	0.675	5.2	3.5	0.418	0.326	normal
86	ND	MARY	50	F	152	49	110	70	81	109	4.5	33.6	8.1	1.536	0.666	11.2	3.9	1.15	0.651	5	3.5	0.375	0.3	normal
87	ND	SANKAR	49	M	166	55	120	80	87	118	4.6	30.8	7.5	1.532	0.756	10.5	3.1	1.33	0.704	4.5	3	0.428	0.333	normal
88	ND	SAHAYAM	58	M	158	59	110	80	102	124	4.1	31.2	7.5	1.538	0.759	10.8	3.8	1.141	0.648	5.2	3.5	0.418	0.325	normal
89	ND	ADHILAKSHMI	65	F	157	58	120	80	98	126	4.8	31.1	7.6	1.525	0.755	10.8	4	1.086	0.629	5	3.6	0.344	0.28	normal
90	ND	RADHA	48	F	166	60	110	70	87	119	4.5	30.5	7.5	1.517	0.754	10.2	3.4	1.201	0.666	5.1	3.5	0.397	0.313	normal

S. no.	Group	NAME	AGE	SEX	Height	Weight	SBP	DBP	FBS	PPBS	HbA1C	OPHTHALMIC ARTERY				CENTRAL RETINAL ARTERY				CENTRAL RETINAL VEIN				FUNDOSCOPY
												OAPSV	OAEDV	OAPI	OARI	CRAPSV	CRAEDV	CRAPI	CRARI	CRVPSV	CRVEDV	CRVPI	CRVRI	
1	DNR	AMULU	36	F	168	75	108	78	128	168	6.9	28.3	7.4	1.456	0.738	9.1	2.3	1.619	0.747	7.6	6.3	0.225	0.165	normal
2	DNR	VALLIYAMMAL	56	F	161	56	118	80	90	118	6.6	39.7	9.2	1.685	0.768	12	5.9	0.504	0.747	12.7	8	0.369	0.509	normal
3	DNR	DURAIRAJ	56	M	156	92	118	80	264	280	7.5	27.8	2.9	2.644	0.894	12.1	3.1	1.34	0.42	9.3	4.2	0.89	0.54	normal
4	DNR	CHINNAMMAL	67	F	162	65	120	80	220	262	7.8	33	6.8	1.767	0.794	14.3	4.3	1.32	0.699	10.8	4.3	1.05	0.606	normal
5	DNR	GANESAN	70	M	165	72	118	86	118	180	6.8	20	3.7	1.83	0.814	8.5	2	1.5	0.761	13.5	3.1	2.3	0.774	normal
6	DNR	SARAVANAN	35	M	168	41	110	80	134	313	7.1	21.2	5.1	1.663	0.758	7.3	2.5	1.051	0.658	5.8	2.4	0.59	0.586	normal
7	DNR	PRASATH	50	M	170	95	112	80	184	322	7.5	30.9	6	1.445	0.805	9.6	2.2	1.732	0.772	9.4	3	1.179	0.684	normal
8	DNR	RAVI	50	M	164	65	120	80	419	508	8.2	45.3	10.5	1.665	0.769	9.4	1.6	1.414	0.829	18.5	9.5	0.834	0.486	NORMAL
9	DNR	MUNUSAMY	57	M	160	64	118	78	287	312	7.5	21.2	4	1.907	0.814	13.9	2.4	1.703	0.826	15.3	6.1	1.034	0.605	normal
10	DNR	GOVINDHASAMY	64	M	173	72	122	80	156	202	7.3	32.4	5.3	1.88	0.838	12.7	2.3	1.799	0.819	14.1	8	0.541	0.431	NORMAL
11	DNR	RADHAKRISHNAN	50	M	159	37	118	80	312	342	8.2	34	6.3	2.048	0.814	6.5	2.1	1.294	0.676	9.4	4.1	0.848	0.566	normal
12	DNR	GNANASAMBANDHAM	51	m	161	47	120	82	206	254	7.2	24.1	3.1	2.284	0.87	10.7	3.8	1.111	0.643	8.5	4.9	0.591	0.416	normal
13	DNR	JAYACHANDRAN	25	M	174	54	118	70	366	402	8.1	26.6	7.1	1.503	0.735	14.4	3.1	1.967	0.785	7.1	3.8	0.78	0.469	normal
14	DNR	VENKATACHALAPATHY	39	M	168	64	120	80	472	512	8	34.6	7	1.761	0.798	18.3	2.6	1.892	0.86	5.6	3.7	0.421	0.344	normal
15	DNR	PATCHAIAPPAN	56	m	163	57	120	60	329	363	7.8	36.9	7.2	1.882	0.804	9.2	2.6	1.207	0.712	7.8	4.8	0.42	0.384	normal
16	DNR	BHARATH	22	M	169	39	110	60	220	284	6.8	28	4.2	2.227	0.85	15.9	6.6	0.892	0.583	5.2	3	0.548	0.423	normal
17	DNR	KUPPAN	55	M	168	49	120	86	225	300	7.2	29.3	5.8	1.549	0.807	28.4	4.5	1.75	0.843	6.7	1.9	1.47	0.722	normal
18	DNR	RAVI	52	M	162	70	118	82	210	292	7.5	31.8	8.8	1.454	0.724	9.1	5.8	0.46	0.367	8.5	2.3	1.27	0.725	normal
19	DNR	KUPPU	58	F	142	75	120	78	302	356	7.9	36.9	7.9	1.655	0.785	13.9	5.8	0.826	0.583	9.7	6	0.516	0.38	normal
20	DNR	UTHRA	25	f	147	64	110	60	234	331	14.7	29.8	6.5	1.681	0.781	9.8	1.6	2.102	0.84	8.7	5.7	0.447	0.341	normal
21	DNR	FATHIMA NOORA	25	F	160	45	110	80	263	284	7.5	33.2	6.1	2.28	0.815	9.8	2.3	1.881	0.767	4.4	3	0.348	0.316	normal
22	DNR	RANI	35	F	144	56	110	80	238	302	7.5	17	4.3	1.505	0.746	14.1	3.9	1.95	0.725	10.2	4.1	1.318	0.595	NORMAL
23	DNR	LAKSHMI	46	F	162	72	110	80	120	156	6.5	22.2	4.6	1.8	0.794	11.1	4.6	0.899	0.587	8.1	4.2	0.769	0.478	normal
24	DNR	SEKAR	55	M	160	52	120	84	422	560	8	16	5.2	1.229	0.675	10.2	2.7	1.445	0.736	6.8	3.3	0.84	0.514	normal
25	DNR	SAGADEVAN	39	M	167	43	110	70	303	368	7.5	27.1	7.4	1.554	0.726	9.7	2.7	1.53	0.716	6.4	3.9	0.587	0.388	normal
26	DNR	SAKTHIDASAN	34	M	162	60	118	80	224	268	7	28.7	6.4	1.858	0.778	10.4	1.9	1.88	0.815	10.9	4	1.229	0.632	normal
27	DNR	RASOOL BEE	30	F	158	72	110	70	147	182	6.9	59.2	11.3	2.2	0.809	5.8	1.4	1.408	0.76	3.5	1.7	0.645	0.5	normal
28	DNR	BHARATHI	42	F	162	73	120	80	195	271	8.5	38.4	10.6	1.371	0.723	7	1	1.858	0.86	5.3	3.9	0.297	0.259	normal
29	DNR	SURESH	56	M	160	58	120	78	180	220	7.5	21.7	6.4	1.381	0.703	12.6	5.1	0.91	0.596	8.4	4.6	0.677	0.447	normal
30	DNR	JAYARAMAN	60	M	159	60	120	78	151	220	7	41.3	8.8	1.739	0.788	15.4	3.9	1.401	0.747	8	5	0.465	0.372	normal
31	DNR	JAYANTHI	60	F	161	68	110	80	149	286	7.1	38.1	8.1	1.73	0.787	16.9	4.9	1.292	0.709	10.2	4.9	0.882	0.591	normal
32	DNR	GANAPATHY	52	M	160	62	120	62	83	110	5.2	30.1	6.4	1.771	0.786	17	3.4	1.559	0.798	7.7	5.2	0.381	0.316	normal
33	DNR	KULANDAIRAJ	65	M	162	58	120	70	183	208	6.8	34.5	6.4	1.705	0.811	16.7	2.4	1.793	0.859	8.4	5.5	0.433	0.344	normal
34	DNR	AMEENA BEE	40	F	161	78	120	70	302	385	7.1	31.9	8.7	1.487	0.727	11.3	2.7	1.349	0.766	6.2	1.8	1.207	0.714	normal
35	DNR	LAKSHMI	47	F	156	65	120	78	225	246	7.3	31.2	8.9	1.326	0.713	4.9	1.8	1.065	0.644	5.6	3.7	0.439	0.348	normal
36	DNR	CHANDRULAKSHMI	45	F	160	58	110	80	208	226	7.6	35.8	5.1	1.987	0.859	17.2	2.7	1.826	0.846	15.1	5.4	1.288	0.642	normal
37	DNR	ABITHA	18	M	165	52	110	60	261	373	7.1	39.9	11.1	1.516	0.722	10.7	2.6	1.183	0.754	6	3.9	0.419	0.344	normal
38	DNR	ARUNA	55	F	162	49	120	80	208	246	7.7	38.6	8.5	1.787	0.78	8.8	3.4	0.901	0.619	5.1	4.5	0.107	0.105	normal
39	DNR	ABIRAMI	60	F	160	58	110	80	202	253	7.5	32.8	8	1.651	0.756	10	3.3	1.19	0.673	4.2	3.1	0.306	0.273	normal
40	DNR	ANNAKILI	47	F	148	49	120	80	266	288	7.5	20	5.3	1.655	0.736	8.5	2	1.477	0.769	3.7	2.9	0.233	0.214	normal
41	DNR	VALARMATHY	50	F	163	61	120	80	283	362	7.3	31.4	7.6	1.91	0.756	9.5	2.5	1.439	0.74	7.6	4.4	0.604	0.425	normal
42	DNR	MANI	60	M	160	56	120	70	280	303	7.5	26.7	4.8	2.095	0.82	13.4	2.6	1.694	0.805	5.4	4.2	0.251	0.226	normal
43	DNR	BALAJI	44	M	172	68	110	70	199	243	7.5	25.1	3.2	2.081	0.871	6.6	1.3	1.918	0.803	4.8	2.2	0.95	0.527	normal
44	DNR	BALAN	50	M	168	70	110	60	200	268	7.1	32.5	5.8	1.837	0.823	4.1	1.2	1.449	0.7	3.3	1.5	0.868	0.542	normal
45	DNR	BABU	55	M	170	66	110	70	213	246	7.3	33	7.1	1.642	0.784	4	1.2	1.359	0.712	4.2	1.8	0.938	0.581	normal
46	DNR	BABY	50	F	168	59	110	60	238	260	7.4	21.7	3.4	1.947	0.845	4.4	1.2	1.401	0.734	4.9	2.2	0.926	0.558	normal

S. no.	Group	NAME	AGE	SEX	Height	Weight	SBP	DBP	FBS	PPBS	HbA1C	OPHTHALMIC ARTERY				CENTRAL RETINAL ARTERY				CENTRAL RETINAL VEIN				FUNDOSCOPY
												OAPSV	OAEDV	OAPI	OARI	CRAPSV	CRAEDV	CRAPI	CRARI	CRVPSV	CRVEDV	CRVPI	CRVRI	
47	DNR	MADAN KUMAR	32	M	160	45	110	70	357	392	7.3	31.3	6	1.922	0.809	15.4	3.8	1.612	0.755	14	4.2	0.823	0.698	normal
48	DNR	MOHAN	38	M	165	50	110	80	303	342	7.4	20.8	4.6	1.856	0.773	13.6	3.8	1.294	0.721	8.7	4.7	0.719	0.462	normal
49	DNR	ANANDHAN	64	M	172	60	110	70	308	392	7.5	30.3	7.5	1.355	0.745	9	2	1.389	0.78	7.9	4.3	0.599	0.458	NORMAL
50	DNR	AMUDHA	60	F	170	62	110	60	302	363	7.8	36.4	6.6	1.388	0.75	11.9	2.8	1.444	0.761	9.7	5.4	0.585	0.44	normal
51	DNR	DHARMAN	60	M	170	60	110	60	318	342	7.3	19.8	5.1	1.444	0.743	18.2	2.8	1.822	0.848	9	4.6	0.674	0.487	normal
52	DNR	DHARANI	60	F	168	59	110	70	323	348	7.2	19.4	3.7	7.694	0.804	19.3	3.8	1.569	0.803	10.1	5.6	0.577	0.45	normal
53	DNR	BHARATH	22	M	170	39	110	80	246	273	6.8	50.2	8.6	1.969	0.828	12.3	2	1.64	0.838	7.8	5	0.281	0.356	normal
54	DNR	BHAVANI	30	F	168	45	110	70	232	264	7.5	32	8.1	1.463	0.745	9.2	2	1.492	0.776	4.1	2.6	0.467	0.367	normal
55	DNR	SHANKAR	48	M	171	82	140	80	273	384	8.1	32.7	9.3	1.318	0.714	13.9	2.7	1.69	0.806	7.5	3.7	0.761	0.5	normal
56	DNR	SAVITHIRI	50	F	170	76	12	80	280	323	7.9	25.7	5.9	1.585	0.769	16.9	3.4	1.739	0.798	6.2	3.7	0.533	0.395	normal
57	DNR	SAROJA	55	F	168	62	120	86	263	338	7.8	17.5	3.4	1.539	0.808	17.8	3.3	1.944	0.817	7	4.1	0.584	0.419	normal
58	DNR	SHANMUGAM	60	M	167	64	120	70	243	282	7.6	38.5	9.6	1.436	0.75	16.6	2.1	2.351	0.872	7.8	2.6	0.364	0.659	normal
59	DNR	VASANTHA	50	F	165	75	120	80	230	256	6.9	34.9	6.9	1.596	0.801	8.8	3.7	0.99	0.581	5.7	4.5	0.215	0.2	normal
60	DNR	VIVEGAN	56	M	169	65	110	70	193	246	7.2	36	12.8	1.163	0.645	8	1.8	1.45	0.775	5.2	3.5	0.387	0.326	normal
61	DNR	MUNUSAMY	56	M	162	61	130	80	260	292	7.4	29.5	8.1	1.789	0.725	8.5	2	1.385	0.766	5.2	3.3	0.432	0.351	normal
62	DNR	MARIAPPAN	60	M	165	64	120	80	243	264	7.3	34.1	8.4	1.436	0.752	8.4	7.3	1.769	0.85	6.1	3.7	0.547	0.397	normal
63	DNR	MARY	65	F	160	58	120	80	193	224	6.9	30.5	8.6	1.253	0.717	9.5	1.7	1.586	0.825	6.9	3.6	0.768	0.478	normal
64	DNR	MANGAMMAL	60	F	162	56	110	70	180	218	6.8	29.9	8.6	1.252	0.713	9.7	1.5	1.625	0.844	6.8	3.7	0.653	0.461	normal
65	DNR	PACHAIYAPPAN	54	M	172	72	100	70	237	283	7.8	32.4	9.6	1.325	0.703	6.2	1.1	1.374	0.826	8	3.2	1.164	0.6	normal
66	DNR	PANDIYAN	60	M	170	68	120	80	232	264	7.6	31.4	8.2	1.433	0.738	10.1	2	1.415	0.804	8.3	3	1.415	0.641	normal
67	DNR	PETCHIYAMMAL	65	F	166	62	110	80	202	246	7.1	29.7	7.1	1.498	0.76	7.1	1	1.735	0.867	5.9	2.5	0.855	0.582	normal
68	DNR	PREMA	65	F	164	65	110	70	164	203	7.2	24.2	4.8	1.632	0.8	6.3	1	1.876	0.849	5.1	2	1.196	0.608	normal

S. no.	S.NO	Group	NAME	AGE	SEX	Height	Weight	SBP	DBP	FBS	PPBS	HbA1C	OPHTHALMIC ARTERY				CENTRAL RETINAL ARTERY				CENTRAL RETINAL VEIN				FUNDOSCOPY
													OAPSV	OAEDV	OAPI	OARI	CRAPSV	CRAEDV	CRAPI	CRARI	CRVPSV	CRVEDV	CRVPI	CRVRI	
1	1	DR	sulochana	47	F	162	68	124	82	156	204	8.8	47.2	8.1	2.25	0.829	11.2	2.2	1.34	0.802	8.1	2	1.869	0.753	MILD
2	2	DR	thulasi	43	F	151	60	120	80	172	255	10.1	56.7	10	1.982	0.823	11.1	2.2	1.348	0.802	6.6	2	1.533	0.696	MILD
3	3	DR	vasanthi	48	F	160	68	110	72	208	256	7.9	47.2	8.1	2.253	0.802	26.6	6.9	1.245	0.739	11.6	6.9	0.492	0.4	MODERATE
4	4	DR	RANI	50	F	158	64	128	88	173	256	7.5	37.6	7.6	2.74	1.204	22.1	7.3	1.345	0.669	6.4	2	1.419	0.687	MILD
5	5	DR	RASHIDA BEGUM	46	F	156	58	110	60	120	230	7.6	30.1	3.2	2.15	0.893	12.9	3	1.52	0.767	7.3	3.2	0.989	0.565	MILD
6	6	DR	NAGALAKSHMI	56	F	165	68	120	76	112	207	7.2	28.7	8	1.228	0.72	16.2	1.7	1.29	0.709	7	1.7	1.814	0.753	MILD
7	7	DR	CHELLAMUTHU	42	m	151	45	114	78	183	253	7.5	32.9	5	2.23	0.848	21	3.2	2.019	0.849	5.1	4.1	0.236	0.207	mild
8	8	DR	BALU	56	M	163	65	110	70	199	264	7.5	38.3	5.4	2.284	0.86	11.6	1	1.472	0.862	8.7	5.1	0.526	0.41	MILD
9	9	DR	VAIRAM	45	F	165	70	112	78	198	226	7.1	31.7	5.2	1.876	0.835	21.3	3.3	2.324	0.845	9.3	6	0.402	0.345	MILD
10	10	DR	GANESAN	65	M	166	68	120	80	201	262	7.1	47	5.1	2.4	0.891	9.9	2.2	1.674	0.778	13.3	5.8	0.981	0.562	MILD
11	11	DR	VADIVEL	60	M	164	76	120	78	301	380	7.8	55.7	5.8	2.99	0.896	9.9	2.2	1.674	0.778	13.3	5.8	0.981	0.562	MODERATE
12	12	DR	RAVIDASS	53	M	165	68	110	80	307	332	9	15.9	2.7	2.348	0.833	14.1	3.2	1.454	0.771	13.9	5.1	1.126	0.63	MODERATE
13	13	DR	RAKESH	18	M	160	60	120	70	252	280	7.8	42.6	4.3	3.356	0.899	17.3	3.9	1.692	0.776	8	5.2	0.432	0.355	MILD
14	14	DR	SRINIVASAN	53	M	170	66	110	80	177	260	7.9	28.7	5.6	1.763	0.803	5.6	1.8	1.045	0.676	5.3	3.2	0.484	0.4	MODERATE
15	15	DR	MALAR	45	F	151	72	120	84	356	446	8.5	33.2	6.1	22.87	0.815	7.1	1.6	1.416	0.778	3.7	2.6	0.332	0.316	SEVERE
16	16	DR	BOMMI	64	F	154	60	110	70	215	297	8.2	18.1	3.7	1.817	0.796	8.8	`	1.631	0.803	7.8	2	1.698	0.743	MODERATE
17	17	DR	SAKUNTHALA	52	f	149	60	120	80	237	288	8	60.2	8	2.294	0.868	9.5	1.9	1.55	0.796	9.7	5.4	0.55	0.44	MODERATE
18	18	DR	MUTHU	50	M	164	53	112	60	187	223	8.2	44.1	13.4	1.335	0.696	11.8	1.8	2.182	0.846	9.7	4.1	1.14	0.58	MILD
19	19	DR	NIRMALA	60	F	156	60	110	80	223	360	8	44.2	8.5	2	0.808	4.2	1.5	1.013	0.65	2.3	1.5	0.426	0.362	MILD
20	20	DR	NANDHINI	45	F	162	54	120	80	228	260	7.9	41.3	5	2.064	0.879	4.4	2.7	0.785	0.529	5.7	2.5	0.885	0.559	MILD
21	21	DR	SOLOMAN	61	m	161	65	110	80	311	385	8.7	30.5	4.9	2.428	0.838	13.4	3.4	1.424	0.75	6.9	3.4	0.812	0.514	MODERATE
22	22	DR	KAMARAJ	55	M	165	58	120	70	196	228	7.9	15.8	2.6	2.194	0.833	15.2	4.2	1.487	0.725	5.6	3.5	0.428	0.37	MILD
23	23	DR	MURUGESAN	65	M	175	58	120	70	306	408	8.9	26.6	3.4	2.275	0.871	7	2.2	1.147	0.692	10.8	5.4	0.833	0.5	MILD
24	24	DR	MANNAR	60	M	170	65	120	60	282	298	9.1	38.7	5.7	2.318	0.854	12.7	2.1	2.681	0.854	8.4	4.6	0.659	0.447	MODERATE
25	25	DR	ANNADURAI	55	M	178	57	120	80	352	392	12.3	23	5.5	1.829	0.761	6.9	2.2	1.15	0.676	6.1	4.9	0.229	0.2	MILD
26	26	DR	ANANDAN	56	M	170	65	110	80	308	382	10	33.9	8.5	1.734	0.75	11.5	1.8	2.275	0.844	9.5	6.1	0.514	0.358	MILD
27	27	DR	JAYARAMAN	78	M	165	62	110	80	124	256	8.2	35.6	6.7	1.821	0.811	11.1	2.8	1.511	0.746	6.4	4.2	0.401	0.333	MILD
28	28	DR	LAKSHMI	60	M	160	68	120	60	270	320	9.5	34	3.1	2.467	0.91	18.3	3.3	1.699	0.822	5.7	3.1	0.534	0.452	MODERATE
29	29	DR	POONGA	60	F	164	52	118	72	302	361	8.5	32	7.4	1.603	0.769	8	2.1	1.317	0.733	5.7	3	0.732	0.469	MILD
30	30	DR	SHANTHI	59	F	147	65	120	72	235	370	7.8	29.4	3.5	2.221	0.882	11.1	2	1.689	0.823	12.4	4.1	1.23	0.669	MILD
31	31	DR	SAROJA	60	F	160	68	110	80	208	252	8.1	33.8	5.1	2.331	0.848	12.2	1.8	1.849	0.855	7.4	5.3	0.334	0.286	MODERATE
32	32	DR	SARAVANAN	37	M	180	65	120	84	438	520	11.5	36.6	5.8	2.2	0.841	12.5	1.9	1.839	0.845	4.5	3.9	0.13	0.12	MODERATE
33	33	DR	SURESH	45	M	168	62	110	80	333	386	9.2	29.2	4.6	2.313	0.841	10	2.5	1.43	0.75	11.5	3	1.8	0.738	MODERATE
34	34	DR	ANNAMALAI	52	M	164	59	110	80	283	306	8.6	18.7	2.9	2.342	0.845	9.6	3.6	0.942	0.621	8	1.6	1.244	0.797	MODERATE
35	35	DR	SULTHAN	68	M	168	70	110	80	364	382	9.2	27.8	8	1.521	0.713	24.1	3.3	2.898	0.857	9.4	4.4	0.944	0.538	MILD
36	36	DR	SABEENA	65	F	165	65	120	70	308	356	8.9	33.5	4.2	2.103	0.874	16.6	2.6	1.788	1.864	6.9	3.7	0.679	0.461	MODERATE
37	37	DR	INBARAJ	48	M	161	51	110	70	370	392	9.3	32.3	4.1	2.3	0.874	6	1.4	1.278	0.765	7.8	3	1.272	0.614	MODERATE
38	38	DR	VELU	33	M	165	78	120	80	303	363	8.8	33.4	5.4	2.306	0.838	21	2.3	2.239	0.89	11.4	6.2	0.702	0.453	MILD
39	39	DR	MAHIMA	45	F	162	58	110	60	246	282	9.1	17.9	4.4	1.591	0.756	10.7	3.7	1.108	0.653	8.5	4.1	0.825	0.513	MILD
40	40	DR	MAHESH	50	M	160	55	110	70	263	285	8.2	29.7	6.2	1.86	0.792	17	3.9	1.483	0.771	11.1	5.2	0.973	0.527	MILD
41	41	DR	ANANTHAKUMAR	58	M	168	65	120	80	320	363	7.5	36.3	6.3	1.697	0.827	14.5	2.9	1.473	0.796	7.6	3.9	0.696	0.481	MILD
42	42	DR	ARIVU	60	F	169	62	110	80	263	340	7.4	23.7	5.6	1.363	0.763	14.6	1.9	1.68	0.868	10.1	3.2	1.381	0.683	MILD
43	43	DR	CHANDRASEKAR	59	M	165	58	110	60	367	428	9.1	28.8	9.3	1.144	0.678	7.2	2.6	1.239	0.642	5.9	3.3	0.542	0.436	MILD
44	44	DR	CHANDRA	55	F	164	60	110	70	363	394	8.9	23.9	7.9	1.132	0.671	6.5	1.5	1.633	0.771	5.3	2.9	0.586	0.456	MILD
45	45	DR	SHANKARAN	60	M	198	58	120	70	326	362	8.5	19.3	2.6	1.85	0.865	4.6	1.1	1.351	0.757	6	3	0.811	0.511	MILD
46	46	DR	CHANDRAN	50	M	169	60	110	80	343	382	8.4	15.8	4.7	1.611	0.704	5.6	1.6	1.264	0.714	6.9	2.7	0.89	0.6	MILD
47	47	DR	DHAMODHARAN	48	M	168	59	120	80	351	376	7.9	15.8	4.7	1.611	0.704	19.4	3.2	1.735	0.833	9.5	4	0.986	0.585	MILD
48	48	DR	DHANUSHKODI	50	M	168	62	110	80	303	343	7.6	16.6	3	1.735	0.82	21.9	3.9	1.671	0.821	9.7	4.4	0.885	0.548	MILD
49	49	DR	GANAPATHY	55	M	170	60	120	80	243	292	7.9	57	6.9	1.845	0.88	10.7	2	1.777	0.816	11.9	4.7	0.886	0.607	MILD
50	50	DR	VARUN	50	M	165	62	120	80	360	392	9.2	31.6	4.1	2.042	0.872	7.9	1.5	1.517	0.804	3.9	2.5	0.422	0.357	MILD
51	51	DR	VASNATA RAJ	51	M	169	79	130	80	245	263	7.8	35.8	9.2	1.473	0.744	14.6	3.9	1.336	0.736	9.5	6.5	0.433	0.319	MILD
52	52	DR	GANESAN	55	M	156	39	110	70	168	232	9	40.5	4.3	1.938	0.893	14.3	1.8	1.459	0.871	15.4	8.1	0.651	0.477	MILD
53	53	DR	GANGADHARAN	60	M	160	45	120	80	170	243	9.3	42.5	4.5	1.928	0.894	12.6	1.8	1.381	0.854	12.9	5.8	0.745	0.549	MILD
54	54	DR	GANDHIMATHY	50	F	165	56	110	70	260	308	7.4	30.4	2.5	1.901	0.918	14.5	1.4	2.058	0.902	15	2.8	1.075	0.811	MILD
55	55	DR	KARPAGAM	50	F	164	54	120	80	246	324	7.3	48.3	4	2.209	0.918	7.4	1.2	1.774	0.835	2.5	1.5	0.533	0.398	MILD

KEY TO MASTER CHART:

SBP –SYSTOLIC BLOOD PRESSURE

DBP-DIASTOLIC BLOOD PRESSURE

FBS-FASTING BLOOD SUGAR

PPBS-POST PRANDIAL BLOOD SUGAR

DR-DIABETIC RETINOPATHY

DNR-DIABETIC WITH NO RETINOPATHY

ND-NON DIABETIC

OAPSV- OPHTHALMIC ARTERY PEAK SYSTOLIC VELOCITY

OAEDV- OPHTHALMIC ARTERY END DIASTOLIC VELOCITY

OAPI- OPHTHALMIC ARTERY PULSATILITY INDEX

OARI- OPHTHALMIC ARTERY RESISTIVITY INDEX

CRAPSV-CENTRAL RETINAL ARTERY PEAK SYSTOLIC VELOCITY

CRAEDV- CENTRAL RETINAL ARTERY END DIASTOLIC VELOCITY

CRAPI- CENTRAL RETINAL ARTERY PULSATILITY INDEX

CRARI- CENTRAL RETINAL ARTERY RESISTIVITY INDEX

CRVPSV-CENTRAL RETINAL VEIN PEAK SYSTOLIC VELOCITY

CRVEDV-CENTRAL RETINAL VEIN END DIASTOLIC VELOCITY

CRVPI- CENTRAL RETINAL VEIN PULSATILITY INDEX

CRVRI- CENTRAL RETINAL VEIN RESISTIVITY INDEX

INSTITUTIONAL ETHICS COMMITTEE
MADRAS MEDICAL COLLEGE, CHENNAI 600 003

EC Reg.No.ECR/270/Inst./TN/2013
Telephone No.044 25305301
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CERTIFICATE OF APPROVAL

To

Dr.M.Thanga Meena
I Year PG in M.D. RD
Department of Radio Diagnosis
Madras Medical College
Chennai 600 003

Dear Dr.M.Thanga Meena,

The Institutional Ethics Committee has considered your request and approved your study titled **"COMPARATIVE STUDY OF ORBITAL DOPPLER PARAMETERS IN DIABETICS WITH RETINOPATHY AND DIABETICS / HEALTHY CONTROLS WITHOUT RETINOPATHY " - NO.29052017**

The following members of Ethics Committee were present in the meeting hold on **02.05.2017** conducted at Madras Medical College, Chennai 3

1.Prof.Dr.C.Rajendran, MD.,	:Chairperson
2.Prof.R.Narayana Babu, MD.,DCH.,Dean, MMC,Ch-3	:Deputy Chairperson
3.Prof.Sudha Seshayyan,MD., Vice Principal,MMC,Ch-3	:Member Secretary
4.Prof.S.Suresh,MS.,Prof.of Surgery,MMC, Ch-3	: Member
5.Prof.S.Mayilvahanan,MD,Director,Inst. of Int.Med,MMC, Ch-3	: Member
6.Tmt.J.Rajalakshmi, JAO,MMC, Ch-3	: Lay Person
7.Thiru S.Govindasamy, BA.,BL,High Court,Chennai	: Lawyer
8.Tmt.Arnold Saulina, MA.,MSW.,	:Social Scientist

We approve the proposal to be conducted in its presented form.

The Institutional Ethics Committee expects to be informed about the progress of the study and SAE occurring in the course of the study, any changes in the protocol and patients information/informed consent and asks to be provided a copy of the final report.

Member Secretary – Ethics Committee

MEMBER SECRETARY
INSTITUTIONAL ETHICS COMMITTEE
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